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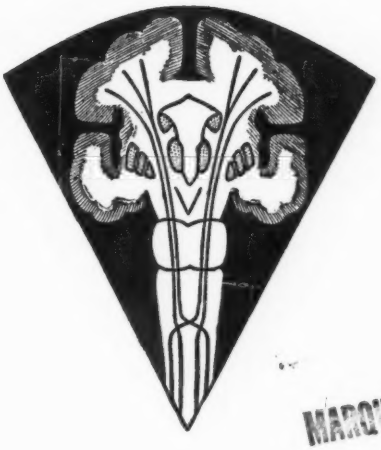

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History

The first investigation of a hemostat with an action comparable to Adrenosem Salicylate was made by Derouaux and Roskam[1] in 1937. They reported that an oxidation product of adrenalin, adrenochrome (which has no sympathomimetic properties), has prompt hemostatic activity.

It was further found that various combinations of adrenochrome, notably the oxime and semicarbazone, produced stable solutions. But, these were so slightly soluble that sufficient concentration could not be obtained for practical therapeutic use. By combining these adrenochrome compounds in a sodium salicylate complex a stable, soluble form can be obtained. This complex has been given the generic name, carbazochrome salicylate, and is supplied under the trade name Adrenosem Salicylate.

Roskam, in his study entitled "The Arrest of Bleeding,"[2] enumerates "the drugs whose efficaciousness as hemostatics have been proved by accurate methods in experimental animals and in healthy men as well. . . . One is the monosemicarbazone of adrenochrome [Adrenosem Salicylate]."

Chemistry

Adrenosem Salicylate is a synthetic chemical. The full chemical name is adrenochrome monosemicarbazone sodium salicylate complex.

Pharmacology

Although it is chemically related to epinephrine, Adrenosem Salicylate has no sympathomimetic effects. It does not alter blood components, nor does it affect blood pressure or cardiac rate.[2-7]

(* U.S. Patent 2,581,850)

Sherber, in an early study,[3] concludes that Adrenosem Salicylate* "is a potent antihemorrhagic factor in those conditions in which the integrity of the smaller vessels is interrupted, and is superior to any similar material that is now available."

He continues, "From our experience it appears that adrenochromazone complex is indicated in preventing vascular accidents incident to hypertension; in maintaining small vessel integrity; in the preoperative preparation where oozing from a vascular bed is anticipated, as in tonsillectomies, adenoidectomies and prostatectomies; and as an adjunct in the treatment of bleeding from such surgical procedures."

Adrenosem Salicylate may be administered simultaneously (but separately) with any type of anesthetic, anticoagulant, or vitamin K and heparin.

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Clinical investigators[2-7] are in agreement that Adrenosem Salicylate controls bleeding and oozing by decreasing capillary permeability and by promoting the retraction of severed capillary ends. It aids in maintaining normal capillary integrity by direct action on the intercellular "cement" in capillary walls. The interesting work of Fulton[8] confirms this. Adrenosem Salicylate, since it is not a vasoconstrictor, has no effect on large severed blood vessels and arterioles.

Adrenosem Salicylate is being used both prophylactically and therapeutically in thousands of hospitals, and in virtually every type of surgical procedure. It has also proved most useful in dental surgery.[7]

Owings reported on the use of Adrenosem Salicylate in controlling postoperative adenoid bleeding in 102 cases.[4] "We have used 2½ mg.

(½ ampule) intramuscularly, 15 minutes before anesthesia for children and 5 mg. (1 ampule) for adults." In only one patient did bleeding occur. Three others showed red blood from the nose and mouth. These patients "were then given 5 mg. intramuscularly, with prompt and complete control. We have also noticed that bleeding stopped more promptly on the operating table."

This is a 1% incidence of postoperative bleeding using Adrenosem Salicylate preoperatively, compared to an incidence of 10% postoperative bleeding in all cases taken from previous records, without Adrenosem Salicylate medication.

Peele reports on the use of Adrenosem Salicylate in treating 178 patients with 24 different conditions.[5] The drug was first used to control postoperative hemorrhage from the adenoid region. He adds: "The results were so dramatic that since that date [1953] Adrenosem Salicylate has been used postoperatively to reduce bleeding from all otolaryngologic and bronchoesophagolic procedures, to treat postoperative hemorrhage from the tonsil and adenoid regions, and to treat selected cases of epistaxis."

The effectiveness of Adrenosem Salicylate in controlling bleeding and oozing in 330 patients is reviewed by Bacala.[6] "Our experience of the effect of carbazochrome salicylate on 317 surgical indications and 13 obstetricogynecological conditions, has been therapeutically encouraging and successful for the control of capillary bleeding. Foremost among the cases studied were 223 tonsillectomies definitely benefited by this metabolic hemostat, making a diminution of the control incidence of posttonsillectomy bleeding of 19.8% down to 7%. It has also been found useful in gastro-intestinal bleeding, cataract extraction, epistaxis, incisional seepage, trans-urethral prostatectomy, menometrorrhagias, cervical ooze, antepartum and postpartum bleeding, threatened abortion, and prevention of capillary hemorrhages during hedulin or dicumerol therapy."

Side Effects

All investigators concur that, at recommended dosage levels, Adrenosem Salicylate is free from toxic effects. No cumulative effects attributable to the drug have been reported.

The only side reaction noted has been a transient stinging sensation in the area of injection when Adrenosem Salicylate is used intramuscularly. As one investigator comments: "The brief discomfort which attends the injection of Adrenosem into the gluteal region has not been a significant problem in children or adults as originally anticipated."[5]

Indications

Idiopathic purpura, retinal hemorrhage, familial telangiectasia, epistaxis, hemoptysis, hematuria.

Postoperative bleeding associated with:
tonsillectomy, adenoidectomy and nasopharynx surgery;
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Dosage

For recommended dosage schedules, please send for detailed literature.

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References

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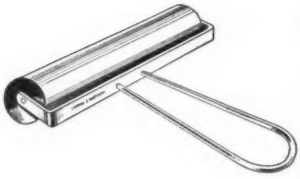

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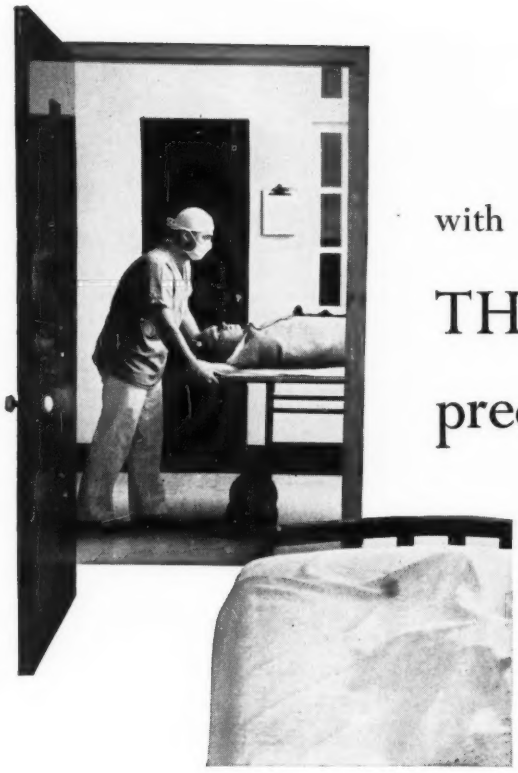

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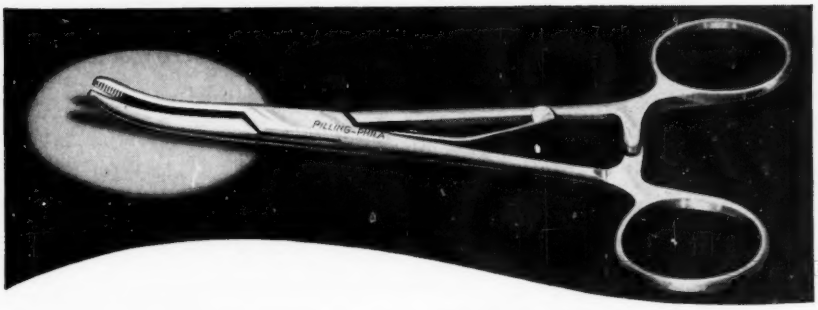

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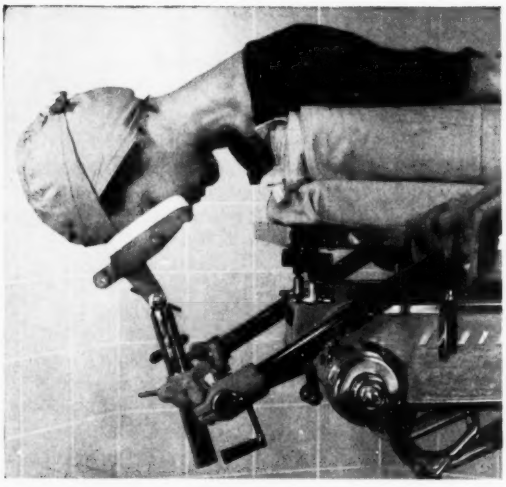

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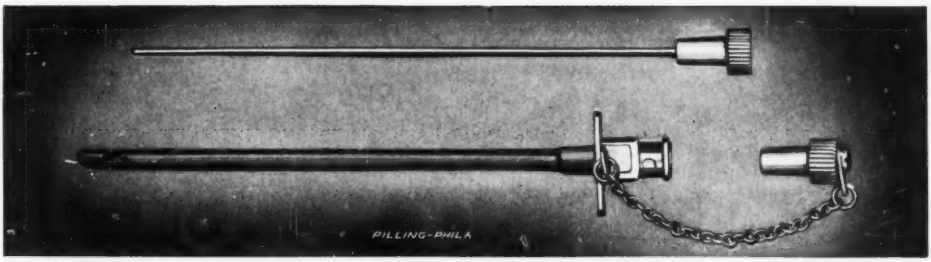

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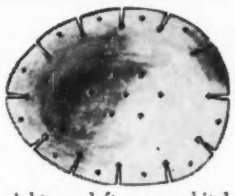

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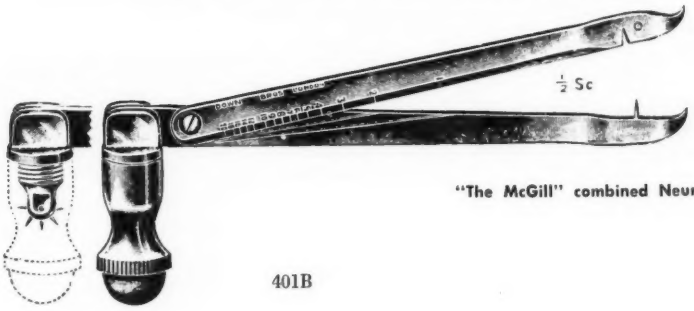

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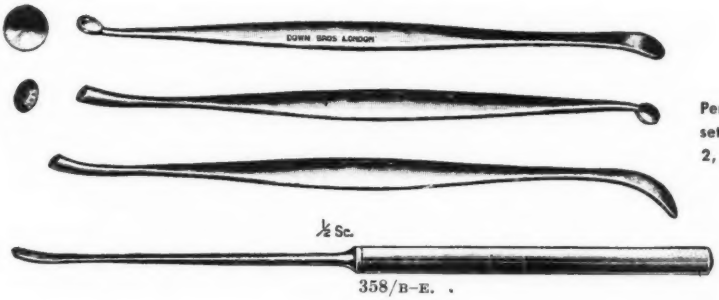

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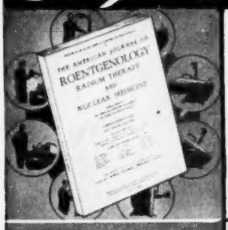

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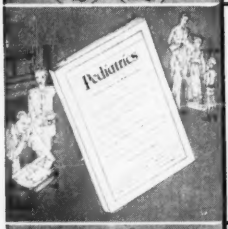

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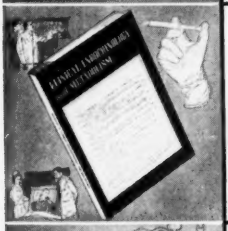

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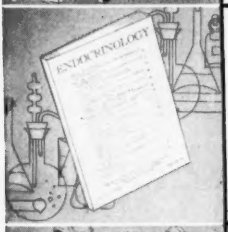

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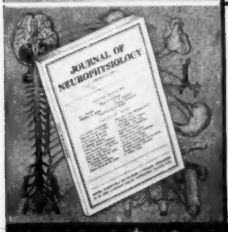

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HYPOTHERMIA, AND INTERRUPTION OF CAROTID, OR CAROTID AND VERTEBRAL CIRCULATION, IN THE SURGICAL MANAGEMENT OF INTRACRANIAL ANEURYSMS

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(Received for publication August 4, 1955)

DISSATISFACTION with the results of operative treatment of aneurysms of the circle of Willis using systemic hypotension followed the study of 19 patients, in whom the aneurysm was attacked directly within 14 days of initial or recurrent haemorrhage. These were not consecutive cases, older patients were not operated on, and there were several examples of serious recurrent haemorrhage. They are, however, not suitable for detailed analysis here, save to note that there were 7 deaths, 4 bad, 1 good and 7 excellent results.[3] This experience led one of us (W.M.L.) to study experimentally methods for protecting the brain from anoxia.

Lougheed and Kahn[13] found impracticable extracorporeal shunts of arterial blood in experimental animals, with or without local cooling. These authors, following the lead of Bigelow, Callaghan and Hopps,[1,2] then assessed general hypothermia and found that substantial protection from anoxia was afforded to the brain in dogs. At 25°C. (77°F.) the cerebral metabolic rate was reduced to 25–35 per cent of normal. Lougheed *et al.*[14] used hypothermia in 2 patients, temporarily occluding both common carotid arteries in the course of a hemispherectomy, and carotid and vertebral arteries bilaterally during the removal of an arteriovenous malformation.

The purpose of this paper is to report our experience of operating on 22 patients at varying periods after rupture of an aneurysm using hypothermia to minimize the effects of anoxia. Temporary occlusion of the great vessels of the neck bilaterally in varying combinations was employed when made necessary by bleeding from the aneurysm or an adjoining artery. These patients were operated on between May, 1954 and April, 1955, and the series includes every case of subarachnoid haemorrhage with an aneurysm demonstrated by bilateral carotid angiography, save one middle cerebral aneurysm operated on successfully. One patient died with an aneurysm of the anterior communicating artery, which was not shown by bilateral angiograms and one other patient who recovered had only one negative angiogram. A large aneurysm was not encountered in this series. These cases were referred either

from the Emergency Department of this hospital or from outlying hospitals to the Neurosurgical and Neurological Services, and this accounts for the wide variation in interval between haemorrhage and operation. Additionally, natural selection eliminates certain bad cases during the period required for diagnosis and transfer.

METHOD

Induction of hypothermia has followed the procedure outlined previously using a bath containing ice water.[13,14] Major changes have evolved in the method of anaesthesia and the control of shivering which seem to make cooling by extracorporeal shunts, as originally described by Delorme,[7] unnecessary.

Pre-operative Assessment. Clinical assessment leading to diagnosis of subarachnoid haemorrhage is followed by electroencephalography and blood studies. Serum potassium, calcium, chlorides, CO_2 combining power, non-protein nitrogen and blood sugar studies are done as often as possible. Percutaneous angiography, usually bilateral, is carried out under local anaesthesia. Assessment of the cardiovascular status of each patient who may undergo hypothermia includes special enquiries of the patient or relatives as to the history, together with clinical examination and electrocardiography. Insufficient emphasis was placed upon this part of the pre-operative assessment in the earlier cases. At this stage the surgeon, anaesthetist, cardiologist and physiologist decide as to the suitability of the patient for hypothermia and the optimum minimum temperature. With increasing age hypothermia is tolerated less well: the older the patient, the more vulnerable is the heart to temperatures below 30°C. (86°F.). In infancy and childhood low temperatures are better tolerated. Any evidence of degenerative cardiovascular disease is an indication that the minimum temperature should be 30°C. (86°F.). Operation using hypothermia is undertaken as soon as it is practical and possible, depending upon the acuteness of the situation and, latterly, in cases of recently ruptured aneurysms it has often been undertaken within 12 to 18 hours following admission.

Anaesthesia. From the beginning moderately large doses of chlorpromazine (Largactil) and promethazine (Phenergan) have been used to produce sedation, to potentiate other analgesics and anaesthetics and to aid in the production of hypothermia. Chlorpromazine (50 mg.) and promethazine (50 mg.) are given intramuscularly the night before operation if the patient is to wait until the following morning, and this is repeated together with Demerol (50 mg.) at six o'clock in the morning. At seven o'clock the patient is taken to the operating room and a slow intravenous drip is begun of 250 cc. normal saline, containing 50 mg. each of chlorpromazine, promethazine and Demerol. The rate of drip is adjusted according to the level of the patient's consciousness. By this time the patient is very drowsy, unaware of his surroundings and easily intubated under topical anaesthesia and light nitrous oxide, trichlorethylene anaesthesia. The gas is administered by continuous high-flow, non-rebreathing, non-CO_2 absorption technique.

In the first 10 cases, Thiopental sodium was used for induction of anaesthesia but it is now omitted. The patient is easily maintained on nitrous oxide with minimal amounts of trichlorethylene. The percentage of oxygen is initially 30 and as the temperature falls it is increased to 60–80 per cent and the trichlorethylene is discontinued. Respirations are voluntary and assistance is rarely added by bagging. Carbon dioxide elimination is carried out by a simple blow-off with 8 to 10 liters per minute of gas flow from the machine.

Electrocardiography. The necessary leads are connected to the patient allowing continuous observations of the electrocardiogram on a fluorescent screen and photographic recordings at appropriate intervals. One lead of the electroencephalograph is used for continuous electrocardiographic recording.

Temperature Recording. A thermocouple is inserted into the rectum and deep in the muscle of the thigh. The temperature of the water bath is similarly recorded until low levels are obtained when a mercury thermometer is used.

Blood Pressure. In the presence of a palpable pulse in the ulnar artery, a Cournand needle is inserted in the radial artery percutaneously and connected with an aneroid manometer.

Electroencephalography. At the conclusion of the cervical dissection the incision in the scalp is marked out and the electroencephalographic leads are placed in the appropriate areas by means of curved needles and the recording is continuous, save for interruptions by the Bovie apparatus.

Cooling of the Patient. Hypothermia is induced in three stages. The first drop in temperature of approximately 1.0°C. (1.8°F.) results from the preoperative sedation with chlorpromazine, promethazine and Demerol. The second stage occurs concomitant with the cervical dissection. During the dissection the patient is in the hypothermic bath on the operating table and his legs, groins and lower abdomen are covered with crushed ice. The extent of the drop in temperature during this stage depends upon the time required for the cervical dissection, which ranges from 20 to 60 minutes. During the third stage the bath is filled with ice water covering the legs and trunk to about the nipple line. In the light of the estimated slide or after-drop in the body temperature which occurs after cooling is terminated, the ice water is removed as the appropriate rectal temperature is approached. The bath may be filled with water at the desired rectal temperature or slightly above it in an attempt to prevent excessive after-drop. Alternatively, the bath may be drained.

Rewarming. When the intracranial procedure is completed, rewarming is begun if the rectal temperature is below 30°C. (86°F.) or if it is above 30°C. (86°F.) and still falling. A limited measure of rewarming is begun by filling the tank with water at approximately 35°C. (95°F.). No active rewarming is carried out if the patient's temperature is steady at 30°C. (86°F.) or has risen to 30°C. (86°F.). The patient is allowed to rewarm himself after

he has reached 30°C. during the surgical closure.

Cervical Dissection and Isolation of Great Vessels of the Neck. The common carotid and vertebral arteries have been defined bilaterally using a transverse incision at the level of the cricoid cartilage. The common carotid artery is defined and retracted laterally. The transverse process of the sixth cervical vertebra can be felt, the apex of the vertebral triangle is identified and in some cases the pulse of the vertebral artery can be felt. The vertebral vein overlies the artery and must be displaced before the artery comes into view. This approach may be complicated by an anomalous vertebral artery. Soft rubber drains are placed around each common carotid and vertebral artery. Should occlusion of these arteries become necessary, a rubber-covered bulldog clamp is placed upon the carotids, and the vertebrals are compressed digitally against the transverse process by an assistant. The common carotid arteries are certainly occluded but it is uncertain if vertebral artery compression is regularly complete.

Bilateral isolation of common carotid and vertebral arteries has been the procedure in 14 cases. In 2 instances the innominate artery was defined on the right and on the left the common carotid artery and the subclavian artery proximal to the vertebral were defined. In 3 cases the common carotid arteries were exposed bilaterally and in 2 cases the internal carotid arteries.

Postoperative Care. The special problems arising from hypothermia during this phase are associated with rewarming and cardiovascular function. The patient is covered only with a sheet and neither nightgown nor pneumonia jacket is worn. The undivided attention of a nurse is required with close medical supervision. Blood pressure, pulse rate, respiratory rate and temperature are recorded at 15-minute intervals. Close watch is kept upon pupils, movement of the extremities and the level of consciousness.

A dangerous degree of systemic hypotension may occur, requiring the intravenous administration of norepinephrine. It may be impossible to puncture superficial veins in the hypothermic patient, so care must be taken to ensure the continued efficiency of an intravenous drip.

Tracheotomy. Coma and evidence of pulmonary atelectasis have been the indications for tracheotomy at the end of the operation and before the endotracheal tube is removed. The development of any respiratory difficulty during the postoperative course has been followed immediately by tracheotomy.

CASE REPORTS

The low mortality and morbidity rate reported by Norlén and Olivecrona[16] following direct surgical attack upon aneurysms 3 weeks or more after rupture has not been achieved by operation in the first 2 weeks. The site of the aneurysm, its size, intracerebral, intraventricular, or subdural clot, arterial spasm and cerebral softening constitute additional factors governing mortality and morbidity. An attempt has been made to record the outcome of surgical treatment of 22 ruptured and 1 unruptured aneu-

rysms using hypothermia and, when necessary, occlusion of the cervical arteries. Our purpose is to allow assessment of the factors bearing on morbidity and mortality in each instance.

Hypothermia has been utilized in 9 additional cases of intracerebral tumours, arteriovenous malformations, etc. without any complications.

Case 1. Rupture of aneurysm of anterior cerebral-anterior communicating complex, left, probably recurrent. Clipping of neck of aneurysm 11 days later: hypothermia and hypotension with bilateral common carotid compression, $7\frac{1}{2}$ minutes. Recovery.

May 10, 1954. L.S., D.60426, a man aged 41, was admitted. In February, 1954, headache suddenly developed with nausea and vomiting. The severe headache gradually subsided over 3 weeks of bed-rest. On May 10, 1954, the patient fell and was found unconscious, breathing stertorously. He slowly regained consciousness, with severe bifrontal headache and vomiting. On examination at that time he was drowsy with a blood pressure of 150/90. No abnormal neurological findings were reported save bilateral extensor plantar response. The cerebrospinal fluid was bloody. Following admission the clinical findings were unchanged, save that only the right plantar response was extensor.

May 11, 1954. Bilateral carotid angiography showed a small (about 0.4 cm. in diameter) aneurysm projecting upwards from the junction of the left anterior cerebral and anterior communicating arteries.

May 21, 1954. Operation using hypothermia.

Hypothermia. The patient was thick-set, fairly obese, weighed 190 lbs. and was 5′8″ in height. The ice water was removed from the bath after $2\frac{1}{2}$ hours when his temperature had dropped from 36.3°C. (97.3°F.) to 31.8°C. (89.2°F.). Over the next $1\frac{1}{4}$ hours he drifted downwards to 28.9°C. (84.0°F.). Irregularities of the nature of auricular fibrillation were noted in the electrocardiogram at 31.8°C. (89.2°F.) and disappeared after some hours in the course of rewarming. The cooling process was otherwise uneventful.

Operation. The aneurysm was exposed between the two frontal lobes by dividing the genu of the corpus callosum. *During the exposure the aneurysm ruptured and both common carotid arteries were occluded for $7\frac{1}{2}$ minutes.* The temperature ranged from 30.7°C. (87.3°F.) to 30.4°C. (86.7°F.) and the mean blood pressure was 58 mm. of mercury. An almost avascular field resulted and a clip was placed across the neck of the aneurysm. The common carotid arteries were released and there was no bleeding. The patient was awake before leaving the operating room when his temperature was 31.0°C. (87.8°F.) and he was talking.

Postoperative Course. His recovery was uneventful. The maximum pyrexia was 38.3°C. (101°F.) without disproportionate tachycardia. There was no neurological deficit. Carotid angiography failed to fill the aneurysm. On June 3, 1954, he was discharged.

Aug. 5, 1954. The electrocardiogram was normal. The electroencephalogram showed irregular activity over the posterior half of the left side of the head. He had returned to his work as a draughtsman and has remained well.

Comment. The marked reduction in bleeding because of bilateral common carotid artery occlusion for $7\frac{1}{2}$ minutes following rupture of the aneurysm allowed adequate exposure of the neck of the aneurysm and accurate application of a clip. An excellent result.

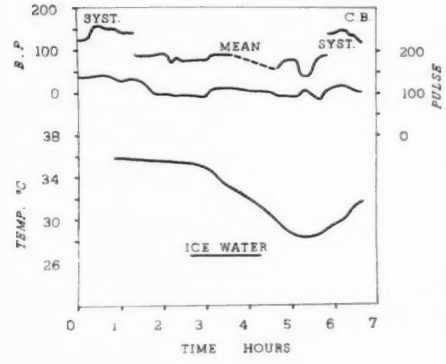

FIG. 1

Case 2. Rupture of aneurysm at bifurcation of left internal carotid. Clipping of neck of aneurysm 44 days later: hypothermia and hypotension. Recovery.

May 17, 1954. C.B., D.60707, a man 34 years of age, was admitted with a history of subarachnoid haemorrhage on April 21, 1954. Neither loss of consciousness nor evidence of focal neurological disorder accompanied the acute ictus. Examination disclosed no neurological abnormalities. Blood pressure was 124/88. He was well.

May 21, 1954. Bilateral carotid angiography showed an aneurysm 2 cm. in length at the bifurcation of the left internal carotid artery.

June 3, 1954. Operation using hypothermia (Fig. 1).

Hypothermia. This man weighed 183 lbs. and was 5′11″ in height. His temperature was 35.4°C. (95.7°F.) when he was placed in ice water in the bath. Over a period of 1 hour and 35 minutes his temperature fell to 31.2°C. (88.2°F.) and the ice water was removed. He then drifted in the next 35 minutes to 29.8°C. (85.6°F.). Warm water was added to the bath, and in the following 35 minutes he reached the low temperature of 29.4°C. (84.9°F.). At the end of the operation his temperature was 31.8°C. (89.2°F.) and he was talking and moving his arms and legs.

Operation. A left transfrontal approach was used and the bifurcation of the left internal carotid was defined. At this stage the mean blood pressure was 80 and the neck of the aneurysm was clipped. The aneurysm was then aspirated when some bleeding resulted. *Arfonad was administered producing a mean blood pressure of 40 and for a minute or two the left common carotid artery was compressed.* Further clips were applied to the aneurysm when it no longer bled.

Postoperative Course. Recovery was without incident and the maximum pyrexia was 38.1°C. (100.6°F.) on the 2nd postoperative day. Tachycardia of 140 occurred the night following operation. A left carotid angiogram showed that the aneurysmal sac did not fill.

June 27, 1954. He was discharged, well.

Comment. Hypothermia provided the assurance that should this aneurysm at the carotid bifurcation have ruptured adequate time and a relatively dry field could be provided by bilateral compression of the common carotid and vertebral arteries. An excellent result.

Case 3. Rupture of aneurysm of anterior communicating artery. Clipping of anterior communicating artery, including aneurysm, bilaterally 4 days later: hypothermia and hypotension: bilateral common carotid occlusion $9\frac{3}{4}$ minutes. Recovery.

June 5, 1954. C.L., D.61691, a 36-year-old man, was admitted to the Emergency Department a few hours after a subarachnoid haemorrhage. He was semicomatose and no neurological signs of localizing value were found. Blood pressure was 128/85.

June 5, 1954. Bilateral carotid angiography showed an aneurysm 1 cm. in diameter occupying the anterior communicating artery and filling only from the right

anterior cerebral.

June 9, 1954. Operation using hypothermia (Fig. 2).

Hypothermia. The patient, weighing 225 lbs. and 5'6" in height, dropped in temperature from 36.4°C. (97.5°F.) to 31.1°C. (88.0°F.) during 2¼ hours' immersion in ice water. For the next 2 hours his body temperature drifted from 31.1°C. (88.0°F.) to 27.1°C. (80.8°F.). At 29.9°C. (85.8°F.) auricular tachycardia occurred and continued intermittently until he was rewarmed. At the end of this procedure he was at 27.3°C. (81.1°F.) and moving his arms and legs.

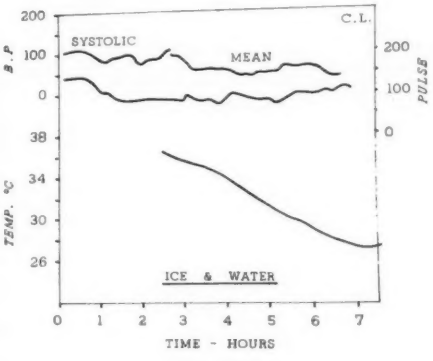

FIG. 2

Operation. Using a right transfrontal approach the brain was found to be very slack. The aneurysm was exposed by separating the frontal lobes and during the exposure the aneurysm ruptured. *Both common carotid arteries were occluded for 2 minutes and 45 seconds while a clip was placed over a hole in the aneurysm.* The carotid arteries were released and dissection was continued. *The clip did not control the bleeding totally and the carotids were again occluded, this time for 9 minutes and 55 seconds.* A relatively dry field was produced and the aneurysm and the anterior communicating artery were completely isolated and clipped on each side. During this procedure the temperature ranged from 28.4°C. (83.1°F.) to 28.0°C. (82.4°F.). Arfonad was started 10 minutes before rupture of the aneurysm, reducing the mean arterial pressure from 64 to 36 mm. of mercury. The combination of Arfonad and haemorrhage reduced the mean pressure further to approximately 30 mm. of mercury. When the aneurysm had been clipped Arfonad was stopped and norepinephrine was started.

Postoperative Course. At midnight of the day after operation he suddenly became unconscious and the bone-flap was reopened. No evidence of intracranial clot was found and the brain was slack. He had two more attacks of unconsciousness of sudden onset, recovering from each within a few hours. Tracheotomy was done to ensure an adequate airway. A right III nerve paralysis developed after the second bout of unconsciousness. Thereafter he improved rapidly and without interruption.

For 3 days his temperature ranged from 39.4°C.–38.9°C. (102.9°F.–102°F.), gradually decreasing to normal by June 18. The pulse rate was 120 the night of operation, reaching a maximum of 140 on June 11.

June 14, 1954. The electrocardiogram was normal.

July 4, 1954. He was well without evidence of focal neurological disorder other than the III nerve paralysis which was recovering, and he was discharged from hospital.

Sept. 15, 1954. There was complete recovery from the III nerve paralysis. The aneurysm was not shown by angiography.

May 24, 1955. He had returned to his former employment. He was well.

Comment. Hypotension and bilateral occlusion of the common carotid

arteries substantially reduced intracranial haemorrhage following rupture of the aneurysm during exposure. Careful dissection of the aneurysm and accurate placing of the clips became possible in spite of rupture of the aneurysm. The postoperative III nerve paralysis remains unexplained. An excellent result.

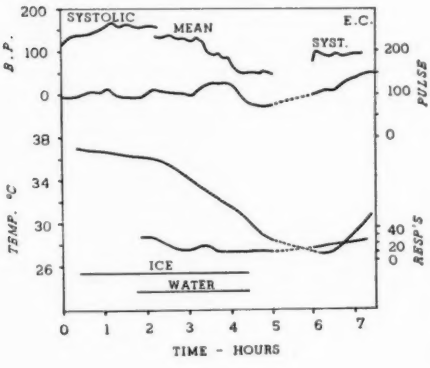

FIG. 3

Case 4. Rupture of aneurysm of left anterior cerebral. Clipping of neck of aneurysm after 25 days: hypothermia and hypotension. Ventricular fibrillation 14 minutes, plus 55 minutes cardiac massage. Recovery.

Aug. 1, 1954. E.C., D.64224, a 50-year-old man, was admitted with a history of subarachnoid haemorrhage on July 10. This was followed by a generalized convulsion. On July 27, 1954 diplopia developed with an internal strabismus. On admission he had questionable early papilloedema and a right VI nerve palsy. Blood pressure was 170/105.

Aug. 2, 1954. Bilateral carotid angiography showed an aneurysm of the left anterior cerebral artery at the level of the anterior communicating, about 1 cm. in length.

Aug. 3, 1954. Electroencephalogram showed a mild dysrhythmia in the right frontal region at 5–7 c./sec.

Aug. 4, 1954. Operation using hypothermia (Fig. 3).

Hypothermia. The patient, weighing 175 lbs. and 5′8″ in height, had ice packed about the groins and legs during the dissection of the neck, and the temperature fell from 37.0°C. (98.6°F.) to 36.2°C. (97.2°F.). During 2 hours and 35 minutes' immersion in ice water his temperature dropped from 36.2°C. (97.2°F.) to 30.4°C. (86.7°F.). Following removal of the ice water there was an additional downward drift to 28.6°C. (83.5°F.). At 33.0°C. (91.4°F.) a tachycardia was noted and at 32.6°C. (90.7°F.) occasional auricular fibrillation occurred. The cardiac rhythm, however, became regular again until 28.8°C. (83.8°F.) when auricular fibrillation recurred and 1 hour and 23 minutes later ventricular fibrillation developed at 28.6°C. (83.5°F.). The mean systolic blood pressure had been 85 and an intravenous drip of Arfonad was started. A rapid drop of blood pressure to 50 occurred and Arfonad was stopped. This was followed by ventricular fibrillation 5 minutes later. After a period of 14 minutes of ventricular fibrillation the chest was opened and cardiac massage was instituted and the heart was continuously bathed in warm water in an attempt to raise its temperature. Chemical defibrillation was attempted without benefit. For 55 minutes cardiac massage was carried out and at the end of this time an electric defibrillator was used with resumption of normal rhythm.

Once the heart recommenced beating no further trouble was experienced. The temperature was 27.0°C. (80.6°F.). During the period of ventricular fibrillation and 55 minutes of cardiac massage spontaneous respirations never ceased although at

one point they were reduced to 2 per minute and continuous electroencephalographic tracing showed that cerebral electrical activity was just visible at the end of an hour with subsequent recovery. During cardiac massage blood pressure in the radial artery could not be recorded with the Cournand needle and aneroid manometer. After the heart continued to beat adequately for some 15 minutes the chest was closed and closure of the bone flap was completed.

At this temperature, 30.8°C. (87.4°F.) corneal reflexes were present.

Operation. Using a left transfrontal approach the brain was found to be slack; the left anterior cerebral artery was defined and as the neck of the aneurysm was being exposed ventricular fibrillation occurred.

Thoracotomy was carried out while the clipping of the aneurysm was completed. No difficulty was encountered in the intracranial part of the procedure and the wound was closed following the resumption of normal cardiac rhythm.

Postoperative Course. As he continued to rewarm, his level of consciousness gradually improved and 2 hours after he left the operating room he was talking in a confused fashion. He was moving his arms and legs. The maximum temperature of 38.7°C. (101.7°F.) with a pulse rate of 110 occurred the night of operation. The day following operation the patient exhibited a Korsakoff psychosis and the findings were in keeping with an acute anoxic encephalopathy. Over a period of 6 days he progressively improved quite rapidly, and more slowly from then on. On his 14th postoperative day the patient's mental status had returned to *almost normal.* He had no complaints. He was then able to remember the names of the attending doctors. He was orientated to time, place and person and the tendency to confabulation had *cleared.* Neurological findings were normal.

Electrocardiogram the day following operation showed a flattening of T.2 and 3, and aVf leads suggestive of myocardial damage. By August 12, the 8th postoperative day, the electrocardiogram was normal.

Aug. 9, 1954. Electroencephalographic studies showed considerable abnormality with slow irregular waves from every region of the head and phase reversals near the tip of the right temporal lobe: a poorly organized alpha rhythm.

Aug. 13, 1954. Improvement had occurred. Frequent waves of 5–7 c./sec. range were more prominent in the left than in the right hemisphere. There was also a little 2–3 c./sec. activity seen irregularly throughout the record.

Aug. 27, 1954. At discharge from hospital the only mental aberration was that of mild euphoria.

Sept. 29–Oct. 2, 1954. He was readmitted for carotid angiography which showed no filling of aneurysmal sac. General and neurological examinations disclosed no abnormality. The patient and his wife considered his mental state to be normal. He is again operating his farm.

Comment. In view of the tachycardia at 33.0°C. (91.4°F.) and the intermittent auricular fibrillation at 32.6°C. (90.7°F.), this patient should not have been cooled below 30.0°C. (86.0°F.). Ventricular fibrillation occurred 5 minutes after Arfonad had produced a sharp drop in blood pressure and was stopped. It appears that the drop in mean systemic blood pressure from 85 to 50 embarrassed coronary circulation of the cold heart, leading to ventricular fibrillation. No studies of blood pH were available. The survival and recovery following 14 minutes of ventricular fibrillation and 55 minutes of

cardiac massage are attributed to the protection afforded to the brain by hypothermia. At the same time, ventricular fibrillation in this case must be attributed to the use of hypothermia. An excellent result, finally.

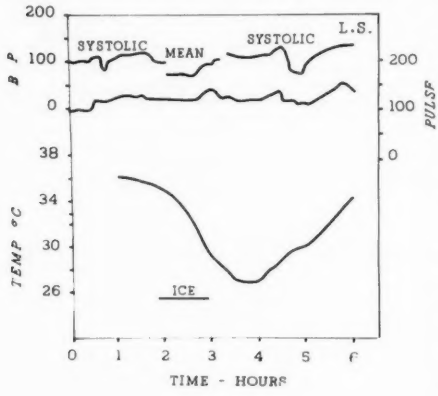

FIG. 4

Case 5. Rupture of aneurysm of right middle cerebral artery. Neck of aneurysm clipped after 22 days: hypothermia and bilateral common carotid and vertebral occlusion 2 minutes. Re-exploration and resection of oedematous frontal lobe. Recovery with motor and sensory deficit left arm.

Aug. 3, 1954. L.S., D.64417, a woman 30 years of age, was admitted to the Toronto General Hospital. She had suffered a subarachnoid haemorrhage on July 28, 1954, without loss of consciousness. Neurological findings were normal save for an equivocal plantar response on the right. Blood pressure was 120/66.

Aug. 3, 1954. Bilateral carotid angiography showed a small saccular aneurysm (5 mm. in diameter) associated with the right anterior cerebral artery. The antero-posterior and oblique views did not demonstrate it consistently. Localization was difficult and one oblique view suggested it was on the right middle cerebral artery. The day following angiography petechial haemorrhages appeared over her face and neck. Her general clinical condition was good and operation was deferred.

Aug. 19, 1954. Operation using hypothermia (Fig. 4).

Hypothermia. She weighed 135 lbs. and was 5′2″ in height. Her temperature was reduced from 36.2°C. (97.2°F.) to 35.8°C. (96.4°F.) by the application of ice around the groins and legs. Following 1 hour of immersion in ice water her temperature had dropped to 30.0°C. (86.0°F.) when the ice water was removed and warm water added. Over the next 50 minutes the temperature continued to drop to 27.0°C. (80.6°F.). During the period of rapid rewarming auricular fibrillation occurred at 29.8°C. (85.6°F.) followed by auricular flutter with a pulse rate of 160. This disappeared as she was warmed. At 34.0°C. (93.2°F.) she was transferred to the recovery room and at this temperature she was moving her right arm and her legs but not the left arm. The maximum temperature was 39.4°C. (102.9°F.) which persisted for 2 days and then gradually subsided.

Operation. A right frontotemporal bone flap was turned. The brain was slack. The bifurcation of the right internal carotid and the beginning of the middle cerebral artery were identified and found to be normal. The right anterior cerebral was inspected by separating the cerebral hemispheres and following the anterior cerebral medially from the bifurcation. In the course of this dissection a small branch of the left (not right) anterior cerebral artery was ruptured. *Both common carotid and vertebral arteries were occluded bilaterally for 2 minutes at 30.2°C. (86.4°F.) while this leak was repaired with a patch of muscle reinforced by Gelfoam.* The mean blood pressure was 90. The right Sylvian fissure was then opened. It was blood, stained and an aneurysm was found 1″ from the bifurcation of the internal carotid. The neck of the aneurysm was clipped.

Postoperative Course. Patient regained consciousness the afternoon of operation but the left arm was paralyzed. Later that night there developed serial focal seizures originating in the middle finger of the left hand. There was no spread to the face. By noon the next day she was becoming drowsy and the right pupil was larger than the left. She had a mild laryngeal stridor.

Operation. On Aug. 20, 1954 the operative wound was reopened. The right frontal lobe was oedematous and tight but was not infarcted. A prefrontal resection of the right frontal lobe was carried out. The dura mater was left open and the bone flap was placed in the deep freeze. The scalp was closed in layers.

A tracheotomy was done.

Postoperative Course. For the following 10 days the patient was drowsy but would answer questions. The left arm was profoundly paralyzed. The seizures in the left hand were controlled with Dilantin, phenobarbital and later by Mysoline.

In mid-September, 1954 the function of the left arm began to return and she made a rapid and striking improvement in power, but with no return of recognition of passive movement, stereognosis or two-point discrimination in the left hand.

Sept. 26, 1954. All movements had returned in the left arm. She had spastic weakness of the left hand with loss of individual finger movements. Cortical sensory deficit persisted. She was discharged.

Oct. 30, 1954. The bone flap was replaced. Her neurological deficit was unchanged.

June 10, 1955. She was well and doing her own housework.

Comment. Localization of the aneurysm was erroneous and handicapped by sensitivity to Diodrast. Manipulation and clipping of parasagittal veins led to swelling of the right frontal lobe to a dangerous degree, requiring frontal lobe resection. Focal seizures of the hand and persisting motor and sensory deficits suggest that either an embolus was dislodged when the aneurysm was clipped or spasm of the middle cerebral artery led to local ischemia and anoxia. Occlusion of cervical vessels did not play an important part. A good result.

Case 6. Rupture of aneurysm of right anterior cerebral artery 2–3 mm. distal to anterior communicating artery. Neck of aneurysm clipped 44 days later: hypothermia and hypotension: bilateral common carotid occlusion 13 minutes. Recovery.

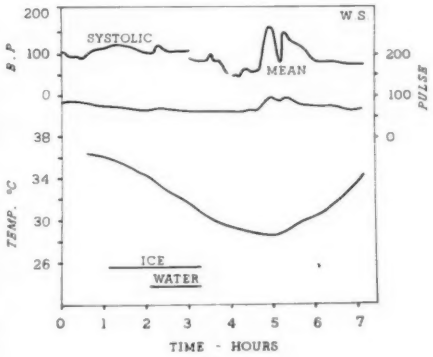

FIG. 5

Aug. 13, 1954. W.S., D.65056, a man aged 47, was admitted to the Neurosurgical Division. On July 13, 1954, while climbing a steel beam 30 feet above the ground a subarachnoid haemorrhage occurred. He did not fall. Lumbar puncture on July 16, 1954, showed blood-stained cerebrospinal fluid. On admission there were no abnormal neurological findings. Blood pressure was 150/90. There was a history of some shortness of breath on exertion. Electro-

cardiogram was abnormal and considered compatible with arteriosclerotic heart disease. The question was raised of an old posterior myocardial infarction.

Aug. 17, 1954. Bilateral carotid angiography showed a small aneurysm, 5 mm. in diameter, associated with the right anterior cerebral artery just distal to the anterior communicating artery.

Aug. 26, 1954. Operation using hypothermia (Fig. 5).

Hypothermia. This patient weighed 150 lbs. and was 5′2″ in height. By packing both groins and legs in ice during the cervical dissection the temperature fell from 36.3°C. (97.3°F.) to 34.0°C. (93.2°F.) in 1½ hours. One hour after the addition of ice and water the temperature was 30.4°C. (86.7°F.). The ice water was removed and 1 hour later the temperature had drifted down to 28.8°C. (83.8°F.). At 29.1°C. (84.4°F.) auricular flutter occurred, changing to auricular fibrillation at 28.8°C. (83.8°F.), and continued intermittently until he was rewarmed to 30.4°C. (86.7°F.) with water at 36.0°C. (96.8°F.). His lowest temperature was 28.6°C. (83.5°F.). He was responding to stimuli at 34.0°C. (93.2°F.).

Operation. A right transfrontal approach was used. Before the dura mater was opened the mean blood pressure was reduced from 80 to 50 by Arfonad. The aneurysm was defined arising from the right anterior cerebral 2–3 mm. distal to the anterior communicating artery and pointing medially and upwards. Additionally, a second small artery, a branch of the right anterior cerebral, entered the aneurysm and emerged from it medially. The neck of the aneurysm was clipped. At this stage the aneurysm was torn and troublesome but no massive haemorrhage occurred. *Bilateral common carotid artery occlusion for 13 minutes, at 29.1°C. (84.4°F.) to 28.8°C. (83.8°F.),* markedly reduced the bleeding and allowed clips to be placed accurately on the small artery, the source of the haemorrhage, passing through the aneurysm. A second source of bleeding was a small perforation in the right anterior cerebral artery where a clip had caught the artery as well as the neck of the aneurysm. This perforation resulted when the clip was teased off to allow accurate placement.

Postoperative Course. Recovery was uneventful. The maximum pyrexia was 38.9°C. (102.0°F.) and the pulse rate was 120.

Comment. Operation upon this anterior cerebral aneurysm, 44 days following rupture, was greatly facilitated by bilateral common carotid compression of 13 minutes' duration and systemic hypotension induced by Arfonad, for the aneurysm was ruptured during operation. An excellent result.

Case 7. Aneurysm of right internal carotid, supraclinoid, unruptured. Clipping of neck of aneurysm 32 days later: hypothermia and hypotension. At 28.6°C. (83.5° F.) ventricular fibrillation, cardiac arrest. Died.

Sept. 9, 1954. B.G., D.66144, a woman aged 58, was admitted to the Neurosurgical Service. For 2 years she had complained of burning right retro-orbital pain and right temporal headache. On Aug. 30, 1954, acute right retro-orbital and right temporal pain developed with dizziness and vomiting, followed by onset of right III nerve paralysis. Examination on admission confirmed the presence of III nerve paralysis. No other significant neurological findings were elicited. Blood pressure was 240/100 with enlargement of the heart and shortness of breath on exercise. The electrocardiogram was reported as within normal limits. The electroencephalogram showed a moderate amount of irregularity and a 5 c./sec. range from many regions of the head.

Sept. 10, 1954. Right carotid angiography showed an aneurysm in the supraclinoid portion of the right internal carotid artery at the level of the origin of the posterior communicating artery. It was 1 cm. in length with a narrow neck.

Oct. 1, 1954. Operation using hypothermia (Fig. 6).

Hypothermia. After immersion in ice water for $1\frac{3}{4}$ hours her temperature had fallen from 36.0°C. to 30.6°C. (96.8°F. to 87.1°F.). The ice water was removed.

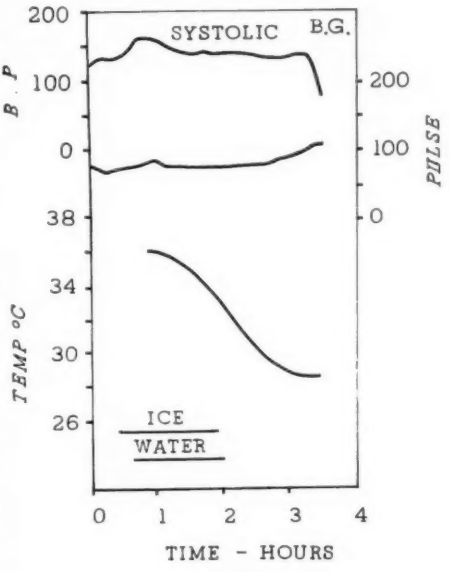

FIG. 6

At 32.2°C. (90.0°F) auricular fibrillation appeared and changed 10 minutes later to auricular tachycardia for 10 minutes which persisted for a further 30 minutes, by which time her temperature was 28.7°C. (83.7°F.). Shortly after this she moved her head, taking a deep inspiration, and Arfonad had just been started. Auricular fibrillation appeared, changing to ventricular fibrillation after a further lapse of 10 minutes, and at a temperature of 28.6°C. (83.5°F.). Arfonad had been stopped just prior to the onset of ventricular fibrillation when her blood pressure was 78/55. Three and one-half hours had elapsed since anaesthesia was begun and 2 hours and 20 minutes since the start of cooling in ice water.

Chemical and electrical defibrillation and cardiac massage failed to restore normal heart action. The myocardium was friable and was torn.

Operation. The aneurysm was approached by means of a subtemporal decompression. The brain was slack with a systolic blood pressure of 140 before Arfonad was administered. The neck of the aneurysm was readily defined and doubly clipped without difficulty.

Neither the common carotid nor vertebral arteries which were exposed bilaterally with tapes around them were compressed.

Autopsy Report. The aneurysm measured 1 cm. in diameter and was not ruptured. The III nerve was adherent to its superior surface and had been compressed. The right optic nerve had been displaced upwards by the aneurysmal mass. Two silver clips had been applied to the base of the aneurysm. The lungs were extremely collapsed and showed marked parenchymal and interstitial haemorrhage of the bases, mostly on the right. This finding was related to cardiac massage as was the tear of the right ventricle.

Comment. Pre-operative assessment in this case was in error. Sufficient emphasis was not placed upon the history of shortness of breath, the hypertension and the large heart. In the light of later experience, it is clear that a minimum temperature of 30.0°C. (86.0°F.) should not have been exceeded and she was cooled excessively. A bad result.

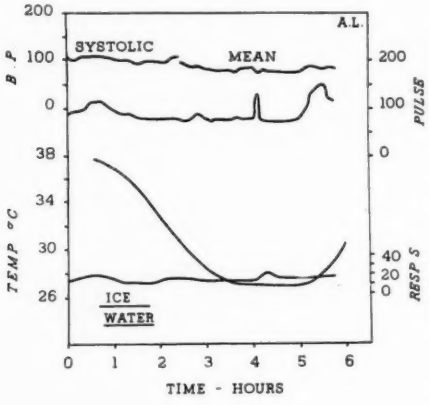

FIG. 7

Case 8. Rupture of aneurysm of right anterior cerebral-anterior communicating artery complex. Aneurysm clipped 4 days later: hypothermia: bilateral internal carotid occlusion 6 minutes. Recovery.

Oct. 14, 1954. A.L., D.67765, a man aged 48, was admitted 10 days following his first severe headache and 4 days following a second episode with verified subarachnoid haemorrhage. He was stuporous. There were no neurological signs of localizing value. Blood pressure was 130/80.

Oct. 14, 1954. Bilateral carotid angiography identified a small aneurysm pointing upwards in the angle formed by right anterior cerebral and anterior communicating arteries.

Oct. 16, 1954. Operation. Both common carotid and vertebral arteries were exposed bilaterally. This dissection proved difficult because of haemorrhage and as a result the intracranial attack on the aneurysm was deferred.

Oct. 18, 1954. Operation using hypothermia (Fig. 7).

Hypothermia. The patient weighed 130 lbs. and was 5′8″ tall with a long thin build. After 1 hour and 10 minutes in ice water his temperature dropped from 37.5°C. (99.5°F.) to 33.7°C. (92.7°F.). The ice and water were removed from the tank and in the next hour and 15 minutes he drifted to 28.4°C. (83.1°F.). From that level further drift was slow, with a drop from 28.4°C. (83.1°F.) to 27.0°C. (80.6°F.) in the following hour.

At the end of the operation the temperature was 32.0°C. (89.6°F.) and he was conscious and talking.

Operation. A right transfrontal approach was utilized. The brain was oedematous and the right frontal lobe was resected in its anteromedial portion for approximately 1½ inches. The anterior cerebral arteries were defined, and an aneurysm 3 mm. in diameter and 4 mm. long was identified lying between the two anterior cerebral arteries as shown by angiography. At that moment the aneurysm ruptured and *both internal carotid arteries were occluded in the neck. Sharp reduction in bleeding was achieved and was further reduced by lightly applying an Olivecrona removable clip to the right anterior cerebral artery.* The neck of the aneurysm was identified and clipped. The clip was then removed from the anterior cerebral and the field was dry. The aneurysm was covered with muscle. The temperature was 27.1°C. (80.8°F.) and the mean blood pressure was 85 during the 6-minute period of occlusion.

Postoperative Course. The day after operation he became confused and disorientated but there was a substantial language difficulty for he was of Central European origin. The confusion cleared by the 3rd postoperative day and the remainder of his course was uneventful. During the 24 hours after operation his pulse rate reached 110 and thereafter it was not above 100. His temperature was 39.4°C. (102.9°F.) for 36 hours, reaching normal on the 3rd day.

Nov. 2, 1954. He was discharged from hospital. Early in January, 1955, he returned to his former work as a nightwatchman.

Mar. 1, 1955. Carotid angiography showed no aneurysm.
May 11, 1955. He was well and working.

Comment. The aneurysm ruptured as it was being exposed and occlusion of the internal carotid arteries bilaterally and the right anterior cerebral artery allowed an excellent view of the region and accurate clipping of the neck of the aneurysm. An excellent result.

Case 9. Rupture of supraclinoid aneurysm of left internal carotid. Evacuation of intracerebral clot in temporal lobe after 13 days. Neck of aneurysm clipped 12 days later under hypothermia: bilateral common carotid and vertebral artery occlusion 11 minutes. Recovery.

Oct. 15, 1954. D.P., D.67910, a woman aged 36, was admitted in a drowsy and dysphasic condition. Positive neurological findings were papilloedema, incomplete paralysis of the left III nerve and left external rectus palsy. Blood pressure was 120/80.

The history indicated that on Oct. 7, 1954, severe headache had been followed shortly by dysphasia. On October 13 a general convulsion occurred shortly after lumbar puncture.

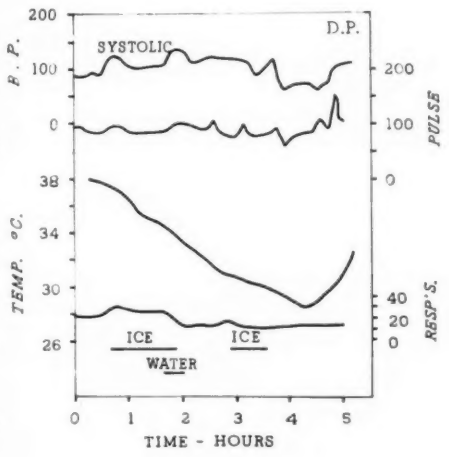

FIG. 8

Oct. 20, 1954. Operation. Ventriculography showed a space-occupying lesion in the anterior half of the left temporal lobe. About 30 cc. of old blood clot were evacuated from this region, using a left temporal bone flap.

Postoperative Course. Recovery was uneventful. The dysphasia and III nerve paralysis were unchanged. She was conscious and her headache was improved.

Oct. 26, 1954. Left carotid angiography demonstrated a supraclinoid carotid aneurysm measuring 1 cm. in length.

Nov. 1, 1954. Operation using hypothermia (Fig. 8).

Hypothermia. She was 5′1″ in height, weighing 90 lbs. Of interest was a drop in temperature from 36.8°C. (98.2°F.) to 34.5°C. (94.1°F.) as a result of packing legs and groins in ice for 1 hour, during the dissection of the neck. Immersion in ice water for 20 minutes depressed her temperature from 34.5°C. (94.1°F.) to 33.5°C. (92.3°F.). A further fall continued following removal of ice and water, for 55 minutes, when her temperature was 31.2°C. (88.2°F.). At this point the aneurysm was coming into view and in order to speed the cooling process the legs and groins were again packed in ice for 40 minutes. The temperature was reduced to 29.8°C. (85.6°F.). During the next 40 minutes the temperature drifted to 28.9°C. (84.0°F.). Cardiac irregularities in the form of auricular flutter appeared at 29.0°C. (84.2°F.) disappearing on rewarming to 32.6°C. (90.7°F.). She was rewarmed with water in the bath at 42.0°–45.0°C. (107.6°–113.0°F.). Her heart rate was 158 and her temperature 33.0°C. (91.4°F.) at the end of the operation. She was not conscious.

The electrocardiogram was normal at the end of the operation.

Operation. Using a left temporal bone flap and a lateral approach, the neck of the aneurysm was defined. In the course of applying a silver clip the neck of the aneurysm was torn almost flush with the wall of the internal carotid artery. Haemorrhage was profuse. The left common carotid was compressed without greatly reducing the bleeding, and then both common carotid and vertebral arteries were compressed. Bleeding from the internal carotid artery was reduced to a trickle. After several attempts successful application of a clip without damage to the internal carotid artery was achieved. *This manoeuvre required 11 minutes during which the common carotid and vertebral arteries were compressed bilaterally with the patient at 30.0°C. (86.0°F.).* Following release of the vessels the systolic blood pressure fell to 65 from 120 and the pulse to 60 from 96. This was of momentary duration.

Postoperative Course. Recovery was uneventful with a maximum pyrexia of 39.0°C. (102.2°F.) the 1st postoperative day, and her pulse rate varied from 80 to 100. She was discharged on the 9th day following operation with a persisting III nerve paralysis.

Mar. 6, 1955. There was complete recovery from the oculomotor paralysis. She was well.

Comment. The relatively dry field resulting from bilateral carotid and vertebral artery compression made possible the technically difficult manoeuvre of applying a clip to the remnant of this aneurysm torn almost flush with the wall of the internal carotid artery. An excellent result.

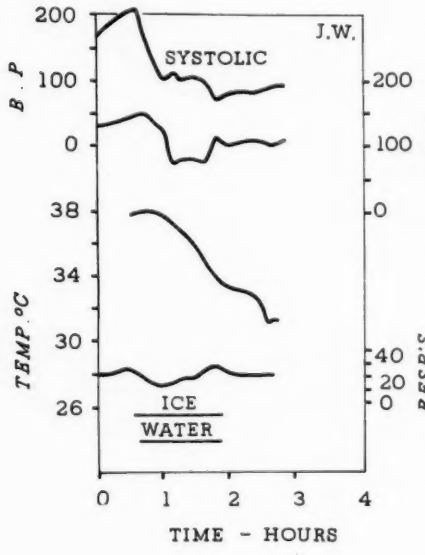

FIG. 9

Case 10. Recurrent rupture of supraclinoid aneurysm, right internal carotid, with intracerebral clot. Evacuation of clot and clipping of neck of aneurysm 4 hours later: hypothermia and hypotension: 10 minutes' unilateral internal carotid artery compression. Died.

Nov. 5, 1954. J.T.W., D.69004, a man aged 41, was admitted. On October 29 he had had an incident suggesting subarachnoid haemorrhage. Disregarding the advice of his physician he returned to work.

On Nov. 5, 1954 a recurrence of headache was accompanied by the development of vomiting and within an hour semicoma. On admission he had a left hemiparesis, with reduction of all forms of sensation. *Two hours following admission, while on the X-ray table awaiting angiography, he had a generalized convulsion with development of bilateral extensor rigidity and he became comatose.*

Bilateral carotid angiography showed a right supraclinoid carotid aneurysm, 1.5×1 cm., at the level of the posterior communicating artery with a large clot in the right temporal lobe.

Four hours later. Operation using hypothermia (Fig. 9).

Hypothermia. Chlorpromazine 50 mg., promethazine 50 mg. and Demerol 100 mg. were given intravenously. The cervical dissection of the right internal carotid artery and the induction of hypothermia in ice water proceeded concurrently. One hour and 20 minutes later his temperature had fallen from 38.0°C. (100.4°F.) to 33.6°C. (92.5°F.) when the ice was removed. After 40 minutes, the temperature was 31.0°C. (87.8° F.). He was not rewarmed and was returned to the recovery room at 31.4°C. (88.5°F.).

At the end of the operation his electrocardiogram showed a wandering pacemaker.

Operation. Through a large subtemporal decompression clot was removed from the right temporal and parietal lobes. Cheyne-Stokes respirations changed to regular breathing. The systolic blood pressure dropped from 207 to 100, and pulse rate from 150 to 74. Hexamethonium and haemorrhage further reduced the systolic pressure to 65. *Fresh bleeding occurred with evacuation of the clot and after a period of 10 minutes of digital compression of the right internal carotid, at 34.6°C. (94.3°F.) the aneurysm was clipped across its neck.* Occlusion of this artery made little difference in the severity of bleeding. The tentorium was divided. At the end of the operation he was still comatose, corneal reflexes were absent, and his extremities were flaccid. Pulse rate 104, blood pressure 88/55, temperature 31.4°C. (88.5°F.).

Postoperative Course. During the night there was no recovery of consciousness. His temperature slid to 28.0°C. (82.4°F.) by midnight. He was then covered with blankets and at 2:30 A.M. his temperature was 29.0°C. (84.2°F.). At 6:30 A.M. hot water bottles were applied to the blankets. This was probably an error of judgment. At 7:30 A.M. his temperature was 29.0°C. (84.2°F.). He died at 8:30 A.M. Between 4 and 7 A.M. his pulse rate rose from 70 to 110.

Autopsy. There was a recent massive subarachnoid haemorrhage extending over the lateral surface of the right cerebral hemisphere and downwards over the pons. Necrotic brain herniated when the dura mater was opened and the temporal lobe was largely destroyed and replaced by a cavity. The brain was very swollen with gross coning through tentorium and foramen magnum. Haemorrhages were present in the mid-brain and pons. Silver clips were in place on the neck of the aneurysm.

Comment. This patient with recurrent haemorrhage and massive intracerebral clot was moribund when surgery and cooling were begun. Extensor rigidity had developed suggesting that coning and brain stem haemorrhages had already occurred. Evacuation of intracerebral clot only prolonged his life. Terminal failure of vital centres was unaccompanied by hyperpyrexia because of induced hypothermia and the use of chlorpromazine.

Case 11. Rupture of aneurysm of right anterior cerebral artery at origin of cingulate branch; aneurysm of right anterior cerebral-anterior communicating artery complex, unruptured. Clipping of neck of distal aneurysm; ligation of proximal aneurysm 7 days later: hypothermia. Recovery.

Nov. 14, 1954. F.F., D.69367, a man aged 49, was admitted in a stuporous condition. Headaches persisting for a week culminated in subarachnoid haemorrhage on November 9 when he lost consciousness. He gradually improved until on November 11 he spoke a few simple phrases and recognized his wife.

Examination revealed a drowsy, stuporous patient unable to execute simple commands. There was early papilloedema. The right arm and leg were rigid and the right plantar response was extensor. He withdrew the arms and legs from pin-

prick. Blood pressure was 145/95.

Nov. 14, 1954. Bilateral carotid angiography showed an aneurysm, 1 cm.×1.5 cm., at the junction of the right anterior cerebral artery and anterior communicating artery and a smaller aneurysm, 3×5 mm., at the junction of the anterior cerebral and cingulate branch.

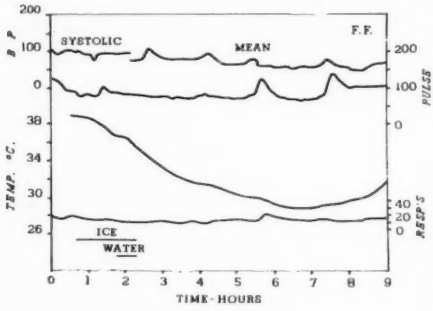

FIG. 10

Nov. 16, 1954. Operation using hypothermia (Fig. 10).

Hypothermia. The patient weighed 175 lbs. and was 6′2″ in height. Body temperature fell from 38.8°C. (101.8°F.) to 37.4°C. (99.3°F.) during the cervical dissection with ice applied to the groins and legs. Immersion in ice water for ½ hour reduced the temperature from 37.4°C. (99.3°F.) to 35.7°C. (96.3°F.). At this stage the ice water was removed and water at 29.9°C. (85.8°F.) was added to the bath. The downward drift of the body temperature continued for 2 hours, reaching 31.5°C. (88.7°F.). From this point he slowly drifted downwards for the following 2 hours, reaching 29.9°C. (85.8°F.). Auricular fibrillation appeared at 30.0°C. (86.0°F.). At the end of the procedure he was moving his arms and legs to painful stimuli, with a temperature of 31.8°C. (89.2°F.).

Operation. A right transfrontal approach was used and the brain was not tight. The exposure was confined to the interhemispheral space and during the exposure of the smaller distal aneurysm at the genu of the corpus callosum, it ruptured. *For a period of about 8½ minutes both common carotid arteries were compressed and for 6½ minutes of this period both vertebrals,* at 29.4°C. (84.9°F.). The haemorrhage was reduced to a point where a small sucker maintained a clear field. A clip was placed across the neck of the aneurysm but encroaching on the anterior cerebral artery. Satisfactory occlusion of the neck of the aneurysm required *two additional periods of occlusion of all four cervical vessels, one of 4 minutes' duration and one of 5 minutes' duration.* During this period an Olivecrona removable clip was applied to the anterior cerebral artery. The second aneurysm was approached without incident and the neck of it was ligated. The mean blood pressure approximated 60 during the periods of occlusion.

Postoperative Course. Immediately after operation the patient moved both arms and legs to painful stimuli, but there was a period of 1 hour of low blood pressure, 70/55. The following day the left side was completely hemiplegic. The left arm and leg were flaccid and there was facial weakness. It seemed clear that thrombosis had developed in the right anterior cerebral artery with retrograde spread to the bifurcation of the internal carotid. The maximum tachycardia was 110 with a temperature of 39.4°C. (102.9°F.) over 4 days.

Jan. 20, 1955. Patient was discharged to his home. He was still markedly retarded mentally. He could answer only simple questions and could feed himself if his food were cut up. There was left lower facial weakness. He could hold his left hand up against gravity or raise it to his face. There was marked spastic paralysis of the left leg. He still required a catheter.

Apr. 18, 1955. He is living in a convalescent nursing home. The left leg remains spastic but he is walking unaided and there is considerably increased usefulness of

the left arm. He carries on an ordinary conversation and reads the daily paper. Urinary incontinence remains a problem.

Comment. This combination of a period of low blood pressure in the immediate postoperative course and operative trauma to the anterior cerebral artery by application of clips predisposed to postoperative thrombosis of the anterior cerebral artery probably with retrograde spread to the bifurcation of the internal carotid artery. A bad result, in spite of continuing slow recovery.

Case 12. Rupture of supraclinoid aneurysm of right internal carotid artery. Neck of aneurysm clipped after 47 days: hypothermia and hypotension: 4½ minutes' bilateral common carotid occlusion. Recovery with cortical sensory deficit of left hand.

Nov. 24, 1954. H.K., D.69917, a woman aged 43, was admitted 28 days after a subarachnoid haemorrhage accompanied by flashes of light and blurred vision. Lumbar puncture in the Sudbury General Hospital demonstrated subarachnoid haemorrhage. She was no longer acutely ill, though complaining of headache. Blood pressure was 120/82.

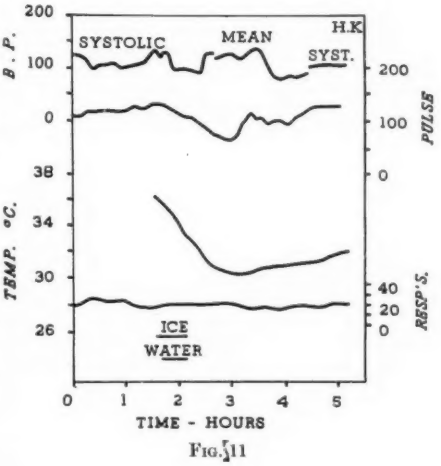

FIG. 11

Dec. 7, 1954. Bilateral carotid angiography showed a carotid aneurysm, supraclinoid and at the level of the posterior communicating artery on the right.

Dec. 12, 1954. Cardiovascular assessment showed consistently normal blood pressures save for the night before operation. The heart was normal in size. The electrocardiogram was abnormal in that there were low T waves in the standard leads and chest leads showed low T waves with a negative T wave in V.3. Dr. Gunton reported: "One might suspect asymptomatic coronary artery disease. She is a suitable case for hypothermia with qualification based on abnormal electrocardiogram."

Dec. 13, 1954. Operation using hypothermia (Fig. 11).

Hypothermia. She was a short obese woman weighing 138 lbs. and measuring 5′2″ in height. After 35 minutes in ice water her temperature had dropped from 36.2°C. (97.2°F.) to 33.2°C. (91.8°F.) and the ice water was removed. Her temperature continued to drop for an additional 40 minutes, reaching 30.6°C. (87.1°F.) where it remained till she was rewarmed by water at 40.0°C. (104.0°F.). She returned to the recovery room with a temperature of 32.7°C. (90.9°F.) recovering consciousness some 8 hours postoperatively, when her temperature was 36.6°C. (97.9°F.). There were no cardiac irregularities during the operation.

Operation. Through a subtemporal approach the neck of the aneurysm was defined without difficulty. Using hexamethonium 15 minutes before the exposure the mean blood pressure was reduced from 120 to 80. As a clip was applied to the neck of the aneurysm bleeding occurred which was thought to be caused by the clip cutting through. *Bilateral common carotid occlusion using bulldog clamps for*

$4\frac{1}{2}$ minutes at 30.8°C. (87.4°F.) produced a relatively dry field. The bleeding point was then seen to be situated on the internal carotid close to the anterior clinoid process where a small branch had been torn. Haemorrhage was controlled by a Gelfoam stamp reinforced with muscle.

Postoperative Course. Recovery of consciousness was uneventful and she was up on the 2nd postoperative day. However, there was quite marked weakness of the left arm with a profound sensory deficit manifest by astereognosis, loss of two-point discrimination, and impaired recognition of passive movement. Motor function, including individual finger movements, recovered, but disturbance of sensation in the hand persisted. At the end of 1 week the electrocardiogram was normal.

Dec. 31, 1954. She was discharged.

Apr. 14, 1955. Recovery has been good for she now cooks, carries her own wood, cleans, and looks after eight children. Headaches accompany menstruation. At times her left hand is stiff. On two occasions in January, 1955, she felt dizzy for a few minutes noting "a shadow coming over right eye and going away."

Visual acuity was 6/6 on the left and 6/7 (corrected) on the right with slight pallor of the right optic disc and peripheral constriction of the field of vision, supporting the presence of optic atrophy. Apart from a cortical type of sensory deficit confined to the second, third and fourth fingers of the left hand, there were no neurological abnormalities. The electroencephalogram was within normal limits. The electrocardiogram showed no abnormality. Blood pressure was 160/90.

Comment. Hypothermia did not prove to be essential in the management of this patient operated upon 7 weeks after rupture of the aneurysm. Identification and control of the bleeding point of the internal carotid was greatly facilitated by systemic hypotension and bilateral occlusion of the common carotid arteries for $4\frac{1}{2}$ minutes producing an almost dry field. It seems probable that the transitory monoplegia and persisting sensory deficit resulted from an embolus dislodged by clipping the aneurysm or spasm of the internal carotid with anoxia. A good result.

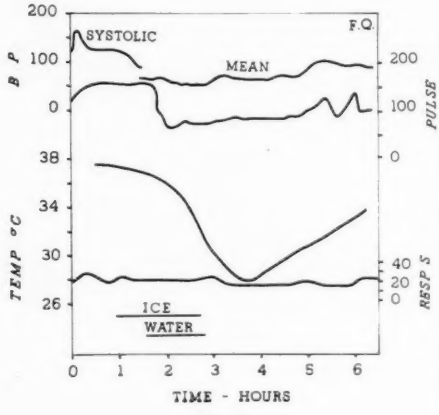

FIG. 12

Case 13. Rupture of supraclinoid aneurysm of left internal carotid. Neck of aneurysm clipped after 36 days: hypothermia: occlusion of innominate artery $4\frac{1}{2}$ minutes, left common carotid and subclavian arteries 6 minutes. Recovery.

Jan. 6, 1955. F.Q., D.71946, a woman aged 36, was admitted. On Dec. 15, 1954 severe pain had developed in the left frontal region behind the left eye, radiating down the side of the nose. This subsided until Dec. 18, 1954, when the pain recurred and on December 22 a left III nerve paralysis developed. At this time bilateral carotid angiography demon-

strated an aneurysm in the supraclinoid portion of the left internal carotid artery at about the level of the posterior communicating artery. Angiography was complicated by a tension pneumothorax.

Examination on admission revealed an apprehensive, young-looking woman with severe left supra-orbital pain, a complete left III nerve palsy, slight right lower facial weakness and slight right hemiparesis. Blood pressure was 130/70. Assessment of the cardiovascular system indicated no significant abnormality. Operation was deferred to allow complete absorption of small pneumothorax.

Jan. 20, 1955. Operation using hypothermia (Fig. 12).

Hypothermia. This woman, 5'3" in height and weighing 100 lbs., cooled quickly and uneventfully. In 1 hour and 20 minutes following immersion in ice water the temperature dropped from 36.8°C. (98.2°F.) to 31.4°C. (88.5°F.) and the ice water was removed. Five minutes later water was added at 30.0°C. (86.0°F.) for her temperature had continued to drop rapidly. During the following 50 minutes while in the bath at 30.0°C. (86.0°F.) her temperature dropped to 28.0°C. (82.4°F.). Rewarming was started ½ hour later by increasing the temperature of the water to 33.0°C. (91.4°F.) and at the end of 2 hours and 15 minutes more she was talking and moving her arms and legs at a temperature of 34.0°C. (93.2°F.).

Auricular fibrillation occurred at 29.0°C. (84.2°F.), clearing on rewarming, and the electrocardiogram was normal at 34.0°C. (93.2°F.).

Tachycardia occurred at 31.6°C. (88.9°F.) and was controlled by 0.5 cc. of 1:2000 Prostigmine given 3 times over a period of 40 minutes.

Operation. Using a left subtemporal decompression the neck of the supraclinoid aneurysm was identified and doubly clipped. The arachnoid was stained with old blood. The corner of the proximal clip perforated the carotid artery. Difficulty was experienced in identifying the site of the bleeding until first the *left common carotid and subclavian arteries were compressed by means of Penrose drains, and then because bleeding was not sufficiently reduced the innominate artery was also compressed.* A practically dry field was obtained and the small perforation was readily identified. Application of muscle reinforced by Gelfoam sealed the perforation, the cervical arteries remaining occluded for approximately a minute more. In all the left common carotid and subclavian arteries were occluded for 6 minutes, and the innominate for 4½ minutes at 29.3°C. (84.7°F.) with a mean blood pressure of 65.

Postoperative Course. Recovery was uneventful but the III nerve paralysis persisted.

Feb. 3, 1955. She was discharged from hospital free of pain.

May 10, 1955. Progressive recovery of III nerve function is taking place. Ptosis has almost completely cleared with diplopia persisting.

Comment. Hypothermia permitted occlusion of the great vessels in the neck expediting accurate identification of the bleeding point on the internal carotid artery and repair of the perforation. Cervical occlusion was not essential. A good result.

Case 14. Rupture of aneurysm at bifurcation of right internal carotid. Clipping of neck of aneurysm and evacuation of subdural hematoma 4 days later: hypothermia. Recovery.

Jan. 10, 1955. D.C., D.72078, a 16-year-old boy, was admitted 2 days after a subarachnoid haemorrhage with loss of consciousness for some hours followed by

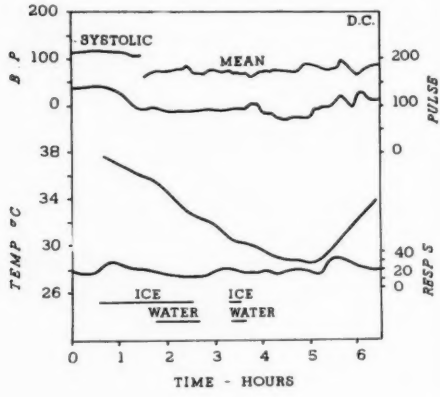

FIG. 13

spastic weakness of the arms and legs. Bilateral extensor responses and patellar and ankle clonus on the left were noted following this episode. Recovery was rapid over the succeeding 9 hours. Cerebrospinal fluid was bloody with a pressure of 350. On admission the lad was alert, orientated and talking. There was moderate increase in activity of the tendon reflexes in the left arm and leg.

Jan. 10, 1955. Bilateral carotid angiography demonstrated an aneurysm, 1.5 cm. × 1 cm., situated at the bifurcation of the right internal carotid artery. Both anterior cerebral arteries filled from the left internal carotid.

Jan. 12, 1955. Operation using hypothermia (Fig. 13).

Hypothermia. The boy weighed 140 lbs. and was 5′11″ in height. Body temperature dropped from 37.6°C. (99.7°F.) to 35.6°C. (96.1°F.) during the hour spent dissecting the innominate artery and left common carotid and subclavian arteries. During this time his groins and legs were packed in ice. After 55 minutes in ice water his temperature was 32.7°C. (90.9°F.) and the ice water was removed. He then drifted to 31.0°C. (87.8°F.) in the next 35 minutes. When the ice and water were replaced for 15 minutes his temperature was further reduced to 30.2°C. (86.4°F.). Over the following $1\frac{1}{4}$ hours he drifted again from 30.2°C. (86.4°F.) to 28.6°C. (83.5°F.). Warm water was added to the bath and at the end of the operation $1\frac{1}{2}$ hours later he was at 33.7°C. (92.7°F.). He was awake, answering questions and moving his arms and legs. At one stage of rewarming, the water added to the bath was too hot and second-degree burns with blisters were produced over the medial aspects of both legs below the knees.

Operation. A right transfrontal approach exposed a subdural clot, about $\frac{1}{4}$″ in thickness, over the right frontal and temporal lobes. The brain was slack. The mean blood pressure was 70. Arising from the bifurcation of the internal carotid was an aneurysm directed forwards and medially and slightly upwards, concealing the anterior cerebral artery which lay posteromedial. The neck of the aneurysm was defined and doubly clipped, sparing the anterior cerebral artery, we thought.

Postoperative Course. Recovery was without neurological incident. Maximum pyrexia was 38.2°C. (100.8°F.) without unusual tachycardia. The second-degree burns were treated with aluminum dust and pressure dressings.

Jan. 25, 1955. Postoperative bilateral angiograms demonstrated no aneurysm and no filling of the right anterior cerebral between the bifurcation of the right internal carotid and the anterior communicating artery. He has remained well.

Comment. The local attack upon this aneurysm at the bifurcation of the internal carotid would not have been attempted without the protection of hypothermia to allow the assistance of cervical occlusion of the great vessels in the neck in the event of operative rupture. Fortunately rupture did not occur. Postoperative angiography indicated occlusion of the right anterior cerebral artery in addition to the neck of the aneurysm. An excellent result.

Case 15. Rupture of aneurysm of right middle cerebral artery at trifurcation: temporal lobe clot. Clipping of neck of aneurysm 36 hours later: hypothermia. Recovery.

Jan. 14, 1955. H.R., W.2927, a 43-year-old male, was admitted 32 hours after a subarachnoid haemorrhage. He was unconscious for 20 minutes following the haemorrhage and in 1–2 hours was observed to be drowsy and confused with a spastic left hemiplegia. On admission it was found that he had deteriorated and he responded only to painful stimuli and was not moving the left arm and leg. Blood pressure was 130/70; pulse rate 40.

Jan. 14, 1955. Bilateral carotid angiography was carried out immediately, demonstrating a small aneurysm in the right middle cerebral artery at the point of trifurcation and a clot in the temporal lobe.

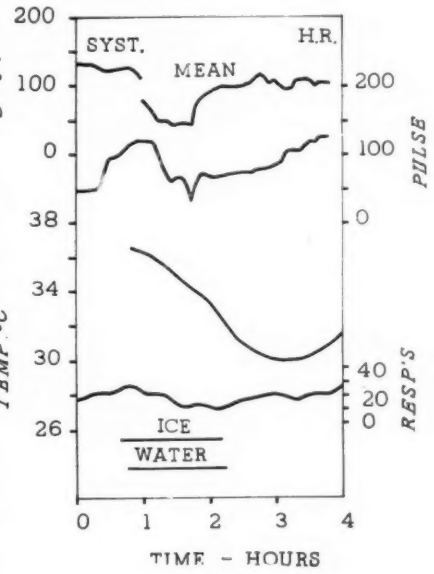

FIG. 14

Jan. 14, 1955. Four hours following admission the patient had deteriorated further and did not respond to painful stimuli.

Jan. 14, 1955. Operation using hypothermia (Fig. 14).

Hypothermia. This man weighed 140 lbs. and was 5′7″ in height. Cooling was rapid and uneventful. During 1½ hours in ice water his temperature had dropped from 36.0°C. (96.8°F.) to 31.9°C. (89.4°F.), when the ice water was removed. A further drift over the next 55 minutes reduced his temperature to 30.0°C. (86.0°F.) at which time warm water was added to the bath. At the close of the operation his temperature was 31.6°C. (88.9°F.), he was moving his right arm and leg spontaneously, and there was withdrawal of the left arm and leg to painful stimulus.

Extrasystoles occurred at 36.0°C. (96.8°F.) after 40 minutes of cooling. Twenty minutes after this bradycardia (36) developed when his temperature was 34.2°C. (93.6°F.) and he was given atropine gr. 1/75. His pulse rate rose to 70.

Operation. Only the right common carotid artery was defined in the neck. A right temporal flap was turned down and the dura mater was found to be very tight. A clot was evacuated from the right temporal lobe. The mean blood pressure was 50. The aneurysm was approached through the Sylvian fissure, not through the clot cavity. Multiple clips were applied to the neck of the aneurysm. The temperature was 31.4°C. (88.5°F.). The mean blood pressure had risen to 100. There was one branch entering the aneurysm proximally and three branches emerged distally. One distal branch supplying the frontal lobe was clipped.

Postoperative Course. His level of consciousness steadily improved. Neither pyrexia nor tachycardia was troublesome. Progressive recovery of power in the left arm and leg occurred over the 10 days following operation.

An electrocardiogram done 24 hours following operation and hypothermia showed flattening of the T waves in all leads.

A right carotid angiogram showed no evidence of the pre-existing aneurysm. The middle cerebral vessels filled. There was no deformity.

Feb. 3, 1955. He was discharged from hospital. He was walking, though there was a persisting mild spastic weakness throughout the left arm and leg with left lower facial weakness. He was still slowed mentally and a persisting disturbance of sensation could not be fully evaluated.

Mar. 18, 1955. He had no complaints. He had recovered from the paralysis. The electrocardiogram was normal. He had returned to work.

Comment. This patient was deteriorating rapidly because of intracerebral clot in the temporal lobe. The common carotid artery was not compressed and hypothermia was not essential, but perhaps afforded some protection to the right cerebral hemisphere during the period of acute compression by swelling and clot. An excellent result.

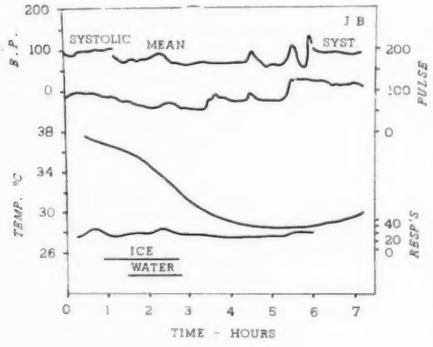

FIG. 15

Case 16. Rupture of aneurysm of left anterior cerebral artery between anterior communicating and bifurcation of internal carotid, recurrent. Operation 29 days after the first haemorrhage, and 24 days after the second haemorrhage: hypothermia. Recovery.

Jan. 26, 1955. J.B., D.73022, a man aged 41, was admitted directly to the Emergency Department. He was found unconscious in a public lavatory. On examination he was semiconscious and restless with a right hemiplegia and bloody spinal fluid. In 4 hours the hemiplegia had completely cleared and bilateral carotid angiography was carried out; this failed to demonstrate convincing evidence of any aneurysm.

Jan. 28, 1955. Electrocardiography showed a sinus rhythm: the Q.R.S. complex showed a slurred R.2–3. The record was judged to be bordering on the abnormal.

Jan. 31, 1955. There was sudden loss of consciousness with development of periodic respirations caused by recurrent haemorrhage.

Bilateral carotid angiography was repeated, as well as vertebral angiography. There were small shadows suggestive of aneurysms on the proximal portions of the anterior and middle cerebral arteries on the left side. These were not considered sufficiently convincing to warrant exploration. Recovery again occurred and his symptoms cleared save for headache.

Feb. 22, 1955. Carotid angiography was carried out on the left side only and showed an aneurysm at the proximal portion of the left anterior cerebral artery, about 1.5 cm. in diameter.

Feb. 24, 1955. Operation using hypothermia (Fig. 15).

Hypothermia. This patient was a somewhat obese male who weighed 180 lbs. and was 5′10½″ in height. After 35 minutes with his thighs and groins packed in ice, the temperature fell from 36.8°C. (98.2°F.) to 36.4°C. (97.7°F.). After 1 hour and 20 minutes of immersion in ice and water a further fall occurred to 32.7°C. (90.9°F.).

At this stage the ice and water were removed. His temperature continued to fall to 28.6°C. (83.5°F.) in 2 hours and 20 minutes, a slide of 4.1°C. (7.4°F.). This slide occurred in spite of the immersion of the patient in water at 31.0°C. (87.8°F.) ½ hour after removal of the ice and water.

There was no significant change in blood pressure. He had hexamethonium and Pronestil again without significant change in the blood pressure, though he was not tilted feet down to any significant degree.

Cardiac irregularities did not occur until the close of the operation when he was in water at 40° C. (104°F.) and being rewarmed. Auricular fibrillation developed at 29.8°C. (85.6°F.) and did not clear up for some hours.

Operation. Using a left transfrontal approach the internal carotid artery was identified. It was displaced to the left and between the internal carotid artery and optic nerve and superior to the optic nerve lay an aneurysm about 1.5 cm. in length and breadth. It was surrounded by thickened blood-stained arachnoid and a layer of clot. The bifurcation of the internal carotid was defined and then the anterior cerebral artery proximal and distal to the neck of the aneurysm. The aneurysm had a wide base and it appeared to be wiser in this case to trap the aneurysm rather than attempt to place a clip across its neck. The fundus of the aneurysm was mobilized so that the aneurysmal sac could be picked up. At this point the junction of the base of the aneurysm and the artery was split and bleeding was profuse. It proved impossible to place clips tangentially along the base of the aneurysm and maintain continuity of the vessel at this point, and the artery was clipped proximal and distal to the aneurysm. Care was taken to avoid dislodging an embolus into the anterior cerebral artery. The aneurysm was opened and allowed to bleed. *These manoeuvres were facilitated by 11 minutes of bilateral common carotid compression at 28.8°C. (83.8°F.).* The mean blood pressure was 70.

Postoperative Course. At the time he was transferred to the recovery room his temperature was 30.1°C. (86.2°F.). He was moving his left leg and arm spontaneously and the right arm and leg only in response to painful stimuli.

During the afternoon following operation his respirations continued at 14. He was fibrillating with a blood pressure of approximately 90/50–60. On return to the ward from the recovery room, he had a sharp drop in blood pressure to 50 mm. or less for 20–30 minutes requiring a cut-down to replace an intravenous needle and further administration of norepinephrine. The night of operation he was moving his arms and legs spontaneously, and he was restless and irrational.

Feb. 25, 1955. Electrocardiogram showed negative T.1, aVl V.5 and V.3. He remained disoriented, forgetful and mentally slow. An extensor response persisted in the right foot.

April 1, 1955. He was discharged to his home.

June 9, 1955. He has shown slow steady improvement in his mental capacity. Wechsler-Bellevue test: fair entering in all faculties, of average intelligence, but tires easily, easily confused, and reduced retention both visual and auditory which interferes with intellectual efficiency.

He had returned to work at a less responsible job than formerly with a view to eventual resumption of draughting.

Comment. Of particular interest is the fact that an aneurysm could not be diagnosed until 29 days after the initial haemorrhage and following the third carotid arteriogram, all of which were technically satisfactory. Hypo-

thermia allowed bilateral common carotid occlusion for 11 minutes, with greatly reduced haemorrhage, and facilitated careful exploration of the aneurysmal sac. The postoperative episode of vasomotor collapse probably produced cerebral hypoxia. The continuing slow improvement in his mental state is impressive. A good result and improving.

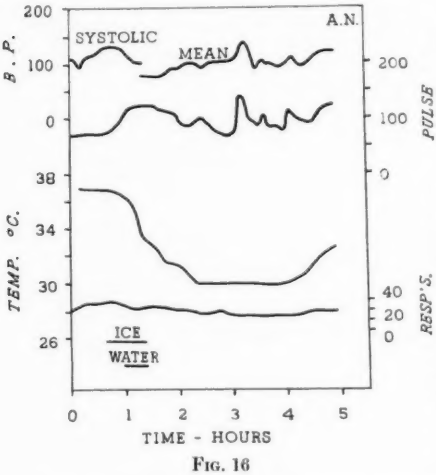

FIG. 16

Case 17. Recurrent subarachnoid haemorrhage: aneurysm of anterior cerebral and anterior communicating arteries. Aneurysm clipped 39 days after second haemorrhage: hypothermia. Recovery.

Feb. 5, 1955. A.N., B.99890, a man aged 52, was admitted. Subarachnoid haemorrhage had occurred in March 1952, and on Jan. 2, 1955. In neither instance was consciousness impaired and there was no neurological deficit. Chronic rheumatic heart disease was present with mitral stenosis and probably also associated aortic insufficiency. Congestive heart failure was not present. The electrocardiogram showed auricular fibrillation. His blood pressure was 150/90. He was digitalized.

Feb. 6, 1955. The electroencephalogram was not remarkable save for a generalized slowing: alpha 8 c./sec. and random 6 and 7 c./sec. waves.

Feb. 7, 1955. Right carotid angiogram indicated an aneurysm at the junction of anterior communicating and anterior cerebral arteries. The left carotid angiogram filled a large 2.5×1 cm. bilocular aneurysm projecting downward from the anterior communicating artery at its junction with the anterior cerebral arteries. This was filled better from the left side than the right.

Feb. 10, 1955. Operation using hypothermia (Fig. 16).

Hypothermia. Packing groins and legs in ice reduced the temperature from 37.0°C. (98.6°F.) to 36.4°C. (97.5°F.) during the 20-minute period required for exposure of both common carotid arteries, and immersion in ice and water dropped the temperature to 33.6°C. (92.5°F.) in 20 minutes. The ice and water were removed and 35 minutes later the temperature had drifted down to 31.0°C. (87.8°F.) when the tank was filled with water at 32.0°C. (89.6°F.). Drift to 30.0° C. (86.0°F.) was slow and the temperature seemed stabilized so that the bath was drained. The lowest temperature was 29.8°C. (85.6°F.). Rewarming was begun $3\frac{3}{4}$ hours following the induction of anaesthesia by adding warm water at 32.0°C. (89.6°F.). He was moving both arms and legs at 32.0°C. (89.6°F.).

Operation. The aneurysm was approached by the left transfrontal route. It proved to be biloculated, lying medial to the left optic nerve and arising from anterior cerebral-anterior communicating complex. The antero-inferior loculus was clipped without reducing its size or tension. The other large loculus, more posteriorly situated, ruptured as the brain was being retracted and bled profusely. *Bilateral compression of common carotid arteries for two periods of 9 minutes and 3*

minutes, at 30.0°C. (86.0°F.) served to reduce the haemorrhage only slightly, so that the sucker barely kept the field clear. The mean blood pressure was 100. Several clips were applied to the aneurysm, sparing the left anterior cerebral artery and controlling the haemorrhage. The right anterior cerebral artery was not seen.

Postoperative Course. He did well with a maximum pyrexia of 38.9°C. (102°F.) on the 2nd and 3rd postoperative days. A left carotid angiogram showed no dye in the aneurysm.

Feb. 23, 1955. He was discharged.

Mar. 4, 1955. He had returned to work. The electrocardiogram was unchanged from the pre-operative record. The electroencephalogram was generally disorganized with some slow features suggesting an epileptic component. The right side showed more abnormality than the left.

Comment. Bilateral common carotid compression reduced only slightly the haemorrhage following operative rupture. The slight reduction was significant in relation to the effectiveness of the sucker. An excellent result.

Case 18. Rupture of aneurysm of right middle cerebral artery at bifurcation: recurrent: intracerebral clot. Aneurysm clipped 35 hours after rupture: hypothermia. Recovery.

Mar. 11, 1955. L.T., D.75403, a male aged 58, was admitted 24 hours following sudden onset of unconsciousness while chopping wood. He remained unconscious for a few minutes. Following this, he was able to walk to his house but by 11.00 P.M. that evening he was stuporous with a left hemiplegia and Cheyne-Stokes respiration.

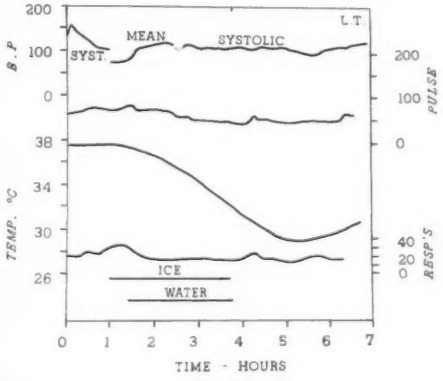

FIG. 17

On admission to hospital, he was drowsy and disorientated, with a left hemiparesis and inattention sensory defect. Blood pressure was 120/74.

Mar. 11, 1955. Bilateral carotid angiograms demonstrated two aneurysms—one at the bifurcation of the right middle cerebral artery, which was a small bulge; the other a pedunculated aneurysm 2 cm. distal to the bifurcation of the right middle cerebral artery, measuring 2.5×2 cm. There was upward displacement of the middle cerebral artery and shift of the anterior cerebral to the opposite side, indicating an intracerebral clot.

Mar. 12, 1955. Operation using hypothermia (Fig. 17).

Hypothermia. The patient weighed 175 lbs. and was 5′11″ in height and of a short obese type. After 2 hours and 25 minutes in ice water the body temperature dropped from 37.2°C. (99.0°F.) to 32.0°C. (89.6°F.). In 45 minutes the patient drifted to 30.0°C. (86.0°F.) and water was placed in the tank at 30.0°C. (86.0°F.). He continued to drift to 28.8°C. (83.8°F.) over the next 40 minutes. Transient auricular arrhythmia occurred at 34.9°C. (94.8°F.) which suggested but was not typical of fibrillation. Thereafter the beat was regular. Rewarming was uneventful.

Operation. Right subtemporal approach. The brain was slack even in the pres-

ence of a large intracerebral clot and the hemisphere was soft and would not have stood much retraction. The clot was evacuated, staying away from the aneurysm. The middle cerebral artery was then identified both proximal and distal to the aneurysm and then the aneurysm was isolated. It was clipped across the base of the neck without rupture. The aneurysm at the bifurcation was but a bulge and was covered with muscle. Spasm of the middle cerebral artery was observed at the end of these manipulations.

Postoperative Course. The patient remained confused and drowsy. On the 2nd day a tracheotomy was carried out because of some respiratory difficulty and slowly over the next 4 or 5 days he made a progressive recovery. On the 6th postoperative day he was walking about the ward and talking rationally.

Apr. 1, 1955. He was discharged from hospital.

Apr. 22, 1955. His only complaint was "whirling" if he extended his neck suddenly. His intellect was unimpaired. Calculation tests showed that he fatigued easily. He had a left lower facial weakness and upper left quadrantic field defect. Otherwise he was normal.

May 17, 1955. He had improved and planned to return to work.

Comment. Hypothermia was not indispensable in this case as the aneurysm did not rupture during exposure and it was unnecessary to shut off the vessels of the neck. However, the damaged brain was protected during the operation from the effects of spasm which occurred in the middle cerebral artery as a result of dissection and clipping of the aneurysm. An excellent result.

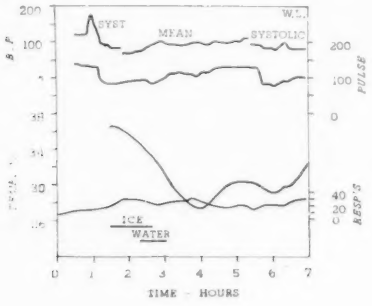

FIG. 18

Case 19. Recurrent rupture of aneurysm of right anterior cerebral-communicating artery complex. Clipping 16 days after last hemorrhage: hypothermia. Recovery.

Mar. 21, 1955. W.L., D.75839, a man aged 42, was admitted. He had had a subarachnoid haemorrhage without loss of consciousness or focal neurological disorder in 1953. On Feb. 22, 1955, a second subarachnoid haemorrhage was followed by semicoma and confusion, clearing in 24 hours. On Mar. 13, 1955, a third haemorrhage occurred without disturbance of consciousness. Neurological findings were normal. Blood pressure was 130/70. There were auricular premature beats recorded in the electrocardiogram.

Mar. 24, 1955. Bilateral carotid angiography suggested an aneurysm associated with anterior communicating artery. This was confirmed by oblique films on Mar. 25, 1955, the aneurysm being situated on anterior communicating artery and filling from the right side only.

Mar. 29, 1955. Operation using hypothermia (Fig. 18).

Hypothermia. The patient was 5′9″ tall and weighed 121 lbs. He was thin. He cooled from 36.8°C. (98.2°F.) to 34.9°C. (94.8°F.) during 45 minutes with legs and groins packed in ice. During 20 minutes in ice water he dropped to 33.4°C. (92.1°F.)

when the ice was removed, and after a further 25 minutes the water was removed and his temperature was 31.2°C. (88.2°F.). His temperature continued to drift reaching 28.4°C. (83.1°F.) in ½ hour. Warm water was added for 20 minutes by which time the temperature was 27.7°C. (81.9°F.). His temperature was then maintained at approximately 30°C. (86.0°F.). by altering the temperature of the water. Auricular fibrillation developed when his temperature had dropped to 29.8°C. (85.6°F.). He continued to fibrillate for 1 hour as his temperature went down to 27.0°C. (80.6°F.) and until it had climbed to 29.2°C. (84.6°F.).

Operation. Both common carotid and vertebral arteries were defined in the neck. Using a transfrontal approach the right anterior cerebral artery was defined crossing the optic tract. Both anterior cerebral arteries were identified with the aneurysm projecting upwards and backwards between them. Before the neck was defined the aneurysm was ruptured. Haemorrhage was remarkably reduced by bilateral common carotid and vertebral artery occlusion, for 8½ minutes at 30.2°C. (86.4°F.) with a mean blood pressure of 90–110, and this period allowed accurate identification and elimination of the aneurysm. The anterior communicating was clipped to the left of the aneurysm, and the neck of the aneurysm and anterior communicating immediately medial to the right anterior cerebral were clipped. The aneurysm was then opened and again bled until additional clips were placed across the fundus and neck. The frontal lobe did not appear much traumatized by retraction and clipping one group of parasagittal veins.

Postoperative Course. Recovery was satisfactory without motor or sensory deficit and he was conscious and talking for the first 36 hours. Then over a 2-hour period he lost consciousness and the pupils became fixed and dilated.

Mar. 31, 1955. Operation. The bone flap was removed and the swollen oedematous right frontal lobe was resected. The arterial blood supply was normal. The dura mater was left open. The bone flap was stored in the bone bank.

Postoperative Course. The right pupil was normal at the end of the operation and 24 hours later the left pupil reacted. He regained consciousness over the ensuing 36–48 hours. From then on recovery was rapid. Tachycardia was a feature the first night. Pyrexia was not troublesome.

Apr. 9, 1955. He was discharged well, save that the bone flap had to be replaced.

May 27–June 1955. He was readmitted. There was no detectable abnormality of cerebral function. Carotid angiography did not fill the aneurysm. The bone flap was replaced.

Comment. Hypothermia and bilateral common carotid and vertebral artery compression for 8½ minutes allowed accurate clipping of the neck of the aneurysm during exposure. Acute cerebral swelling postoperatively required frontal lobe resection. An excellent result.

Case 20. Rupture of aneurysm of anterior communicating artery: reaction to Diodrast. Clipping of aneurysm 2 days later: hypothermia. Died.

Mar. 29, 1955. I.H., W.4216, a woman aged 50, was admitted. She had been known to have hypertension for 4 years. On Mar. 28, 1955, subarachnoid haemorrhage occurred without loss of consciousness. On March 29 diplopia and subconjunctival haemorrhages were observed. Blood pressure was 190/110.

Mar. 30, 1955. Bilateral carotid angiography showed an aneurysm, 1 cm. in diameter, of the anterior communicating artery filling from both anterior cerebral arteries. Following injection of Diodrast she became comatose for a few minutes and

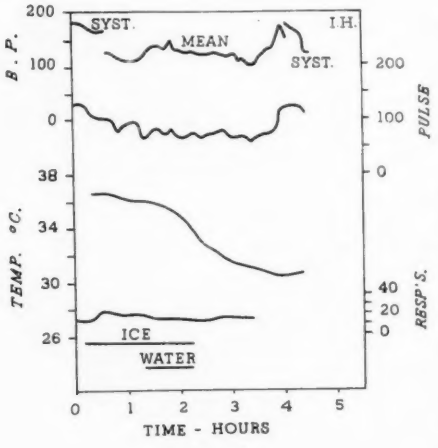

FIG. 19

a profound left hemiplegia developed, more marked in the face and arm than in the leg.

Mar. 30, 1955. Operation using hypothermia—2½ hours following arteriogram and onset of hemiplegia (Fig. 19).

Hypothermia. This woman weighed 125 lbs. and was 5'3" in height. The temperature fell from 36.8°C. (98.2°F.) to 36.1°C. (97.0°F.) during 1 hour and 10 minutes when her legs and groins were covered with ice. For the next 55 minutes the bath was filled with ice water and her temperature dropped to 33.8°C. (92.8°F.). The ice water was removed and over 1 hour and 40 minutes her temperature drifted to 30.8°C. (87.4°F.), the low temperature.

No cardiac arrhythmia developed. Tachycardia became a feature at 30.8°C. (87.4°F.).

Operation. Common carotid and vertebral arteries were defined bilaterally. A right transfrontal approach was used. Because of the oedema of the frontal lobe it was not possible to expose the proximal portion of the right anterior cerebral artery. An aneurysm occupying the anterior communicating artery was identified and occluded, using two Olivecrona clips. A small amount of old clot extruded from the frontal lobe, under the tip of the retractor, probably from the region of the basal ganglia. The dura mater was left open and the bone flap was removed.

Postoperative Course. She moved her right arm and both legs. The day after operation she was mumbling a few words and moving both arms and legs, the left less well than the right.

Apr. 1, 1955. She was brighter, talking better, and power was improved in the left arm.

Apr. 2, 1955. During the morning she was drinking and appeared to be progressing favourably. Over a period of ½ hour she became comatose. The pupils were very small and unreactive to light. She had a left flaccid hemiplegia. Hypertension persisted at 200/110. The pulse rate was 70. The respirations were periodic.

Apr. 2, 1955. Operation. The scalp flap was turned down. The frontal lobe was extremely swollen and tight, and had herniated through the bone defect so that it protruded about ⅜". The frontal lobe was resected. The superficial arteries bled as did the veins. No clot was seen and the ventricle was tapped without encountering clot. The dura mater was left open. The scalp was closed.

Apr. 15, 1955. The patient had not recovered consciousness. Bilateral extensor rigidity of legs became marked in response to stimulation and the arms extended and pronated.

She remained at a vegetative level until her death on June 2, 1955.

Autopsy. The clips were on the aneurysm and anterior communicating artery and did not encroach upon either anterior cerebral artery. Thrombosis had occurred in both anterior cerebral arteries with extensive cerebral softening.

Comment. The degree of success attending operation in the preceding

cases led to increased willingness to accept greater risks in the form of arteriosclerosis and hypertension. In retrospect an error of judgment was made for the oedematous frontal lobe should have been resected initially and a generous subtemporal decompression provided in addition to leaving the dura mater open and removing the bone flap. A bad result.

Case 21. Rupture of right middle cerebral aneurysm, probably recurrent. Ligation of neck of aneurysm 5 days after first haemorrhage: hypothermia. Died.

Mar. 31, 1955. I.D., D.76270, a 50-year-old woman, was admitted. She was a known diabetic requiring insulin and was hypertensive. On Mar. 26, 1955 she had suffered from severe headache followed by drowsiness and confusion. The spinal fluid was bloody. On March 29 she became more drowsy and confused. The night of March 30 her left leg became weak, and on March 31, the day of admission, her level of consciousness deteriorated further.

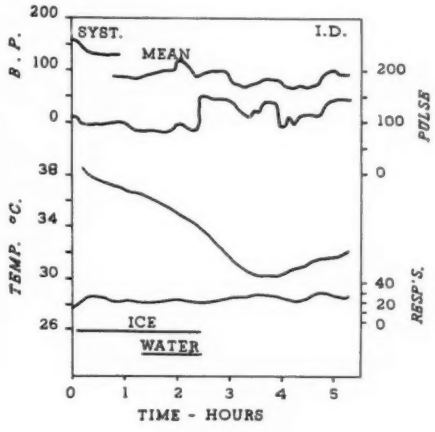

FIG. 20

On examination she was drowsy, disorientated, and her neck was rigid. The pulse rate was 80, with a blood pressure of 220/100. The heart was enlarged but there were no signs of failure. There was a left hemiparesis with a left upgoing toe. The electroencephalogram was suggestive of a right temporal lobe lesion. The electrocardiogram showed left axis deviation. Blood sugar was 155 mg. per cent; CO_2 combining power 44 vol. per cent; urine: sugar 3+, acetone 3+, albumen 2+.

Mar. 31, 1955. Bilateral carotid angiograms demonstrated a right middle cerebral aneurysm about 1 cm. in diameter at the main division of the artery.

Apr. 1, 1955. Operation using hypothermia (Fig. 20).

Hypothermia. This patient was obese and short, weighing 115 lbs. The temperature dropped from 38.4°C. (101.1°F.) to 36.3°C. (97.3°F.) in 1 hour and 25 minutes after the legs and groins were packed in ice. Water was added to the ice and in 1 hour and 10 minutes the temperature reached 33.6°C. (92.5°F.). Water at 30.0°C. (86.0°F.) was added to the tank and after 1 hour the body temperature had drifted to 30.3°C. (86.5°F.) and was stabilized there. Auricular tachycardia occurred early in the course of cooling at 34.0°C. (93.2°F.). At 30.4°C. (86.7°F.) ectopic beats developed and auricular fibrillation began at 30.3°C. (86.5°F.). She was rewarmed and at 32.2°C. (90.0°F.) she was moving both right and left extremities voluntarily but was not conscious.

Operation. Right temporal bone flap. The Sylvian fissure was opened distal to the aneurysm. The aneurysm was easily identified; the neck of it was dissected and ligature was tied around it. There was no rupture or intracerebral clot. There was some brain swelling. The bone flap was replaced and a small decompression was left.

Postoperative Course. At 6 P.M. the night of operation she was moving her arms and legs but was not conscious. During the night her temperature rose to 39.5°C. (103°F.) and she died at 5 A.M.

Autopsy. There was massive swelling of the right cerebral hemisphere. Cerebral softening in the distribution of the right middle cerebral artery was judged to be of more than 24 hours' duration. The right middle cerebral artery was patent but narrowed immediately distal to the aneurysm by an atheromatous plaque. There was no intracerebral clot. Tentorial coning was present and there were brain stem haemorrhages.

Comment. It seems probable that the hemiparesis developing 36 hours after the subarachnoid haemorrhage was caused by atheroma and spasm of the middle cerebral artery resulting in occlusion and cerebral softening. Removal of the bone flap initially and a large subtemporal decompression might have compensated for the cerebral swelling. A bad result.

Case 22. Recurrent rupture of supraclinoid aneurysm of left internal carotid artery directed forwards. Neck of aneurysm clipped: hypothermia. Recovery.

Apr. 2, 1955. M.McG., D.76456, a male aged 26, was admitted. He had suffered a subarachnoid haemorrhage on Oct. 16, 1953, without coma or focal neurological disorder. A similar episode occurred on Mar. 19, 1955. Carotid angiography at the Kingston General Hospital indicated an aneurysm pointing forwards and upwards at the level of the uppermost portion of left internal carotid.

Apr. 5, 1955. He became confused and dysphasic with severe headache, weakness of right face and arm, and hyperactive tendon jerks in the right arm. Recheck angiography showed this aneurysm (2×0.7 cm.) to be arising immediately below and in front of the bifurcation of the left internal carotid.

Apr. 6, 1955. Operation using hypothermia.

Hypothermia. This man weighed about 150 lbs. and was about 5′11″ in height. After 40 minutes in ice the temperature dropped from 38.0°C. (100.4°F.) to 36.5°C. (97.7°F.) and after 50 minutes in ice water the temperature was 33.2°C. (91.8°F.). He continued to drift down to 29.8°C. (85.6°F.) during the hour following removal of ice and water. His temperature then ranged between 29.8°–29.4°C. (85.6°–84.9°F.) for the next 2 hours. Warming was without incident.

The electrocardiogram was normal throughout cooling.

Operation. The left internal carotid artery was exposed by subfrontal approach. Old blood clot extruded from the inferior surface of the left frontal lobe. About 25 cc. of clot was sucked out. The neck of the aneurysm was identified emerging from the anterolateral aspect of the internal carotid immediately below the bifurcation. In spite of a clip occluding $\frac{2}{3}$ of the neck of this aneurysm, bleeding was profuse when the fundus was mobilized and ruptured. Bilateral common carotid artery occlusion for $13\frac{1}{2}$ minutes at 29.6°C. (85.3°F.) did little to reduce haemorrhage and in error the vertebral arteries were not occluded; the aneurysm was collapsed by multiple clips. At this time the internal carotid artery was in spasm and reduced to about $\frac{1}{3}$ of its original diameter. A bleeding point on the middle cerebral was controlled by application of Gelfoam. Finally good pulsation was observed in the internal carotid with lessening of the spasm.

A prefrontal lobectomy was performed and the wound was closed with a generous subtemporal decompression.

Postoperative Course. On April 7, 1955 he was conscious with good power in the right arm and leg. He was not talking.

Apr. 8, 1955. Right hemiparesis and facial weakness became evident with improving level of consciousness.

Apr. 9, 1955. A profound right hemiplegia with marked facial weakness and global aphasia was present. He was fully conscious.

Apr. 13, 1955. The hemiplegia was becoming spastic and he was attempting to speak.

May 7, 1955. He was discharged home. Nominal aphasia was present which was severe enough to prevent him from speaking fluently but he was able to express himself using circumlocution. He had right facial weakness and right spastic hemiparesis, most marked in the leg. The power in the arm and thigh muscles was 4–5[15] and in the ankle 1–2. Sensory system was intact. Individual finger movements were present.

June 9, 1955. Considerable improvement has occurred. Mild nominal dysphasia remains. The right spastic hemiparesis persists, most marked in the leg. There is no sensory deficit. He is doing odd jobs on a farm.

Comment. The full advantage of hypothermia was not utilized as the vertebrals were not occluded. This made the elimination of the aneurysm more difficult. It appears that spasm of the internal carotid arterial tree recurred postoperatively with the development of a limited and as yet undefined amount of cerebral softening. A fair result to date.

Case 23. Recurrent subarachnoid haemorrhage; aneurysm of left middle cerebral artery. Clipping of neck of aneurysm: left anterior temporal lobectomy: hypothermia. Recovery.

Apr. 5, 1955. B.W., D.76549, a woman aged 40, was admitted. Subarachnoid haemorrhage had occurred on Mar. 31, 1955. when she was unconscious for ½ hour. On recovering consciousness she was confused, disorientated and aphasic. The aphasia improved for 3 days but recurred on April 4, with increasing drowsiness and headache.

On admission, 5 days after bleeding, she was conscious and alert, with a nominal aphasia, some headache, slight stiffness of her neck, right hemianopsia and right hemiparesis, but no demonstrable sensory deficit.

April 6, 1955. Following transfer to the X-ray Department for arteriography, she became comatose and hemiplegic. An arteriogram was carried out and showed an aneurysm, 0.5×0.7 cm., at the first main division of the left middle cerebral artery, with evidence of an intracerebral clot in the left temporal lobe.

During a period of 1 hour while she was being prepared for operation, her aphasia remained the same and she remained totally hemiplegic but her level of consciousness improved.

Apr. 6, 1955. Operation using hypothermia.

Hypothermia. This patient was 5′0″ in height, weighed 130 lbs. and was obese. Immersion in ice water for 1 hour reduced her temperature from 36.6°C. (97.9°F.) to 32.0°C. (89.6°F.). Over the next 45 minutes her temperature dropped to 29.4°C. (84.9°F.). When the ice was removed warm water was added to the bath. Her temperature remained stable at 29.4°C. (84.9°F.) for the next 45 minutes and then gradually climbed until it reached 31.4°C. (88.5°F.) 1 hour and 25 minutes later. No difficulties were encountered in the cooling or rewarming process. At the end of operation at 31.4°C. (88.5°F.) she was moving both arms and legs. Auricular fibrillation was present for a few minutes when her temperature was 30.5°C. (86.9°F.). Systolic blood pressure dropped from 100 to 60. During the early phases of cooling this drop in pressure was corrected by the use of norepinephrine intravenously. At

29.6°C. (85.3°F.) auricular fibrillation occurred again.

Operation. Through a left temporal bone flap the Sylvian fissure was opened up, disclosing the aneurysm. The clot, perhaps 15 cc. in all, was evacuated from the fissure and adjoining temporal lobe. The aneurysm was then dissected out and several clips were applied to its neck. During this phase there was bleeding from the fundus of the aneurysm but this was not profuse and did not require occlusion of the vessels of the neck. The anterior 5 cm. of the temporal lobe were resected. A generous subtemporal decompression was left.

Postoperative Course. Maximum temperature was 37.2°C. (99.0°F.) per rectum. The patient was alert but was totally aphasic with a right hemiplegia as before operation.

May 19, 1955. She was transferred to Lyndhurst Lodge for rehabilitation.

Progress Note. She remains totally aphasic with a right hemiplegia. She is bright and up walking around the ward.

Comment. The intracerebral clot appeared old and neither by virtue of its size nor situation accounted satisfactorily for the onset of hemiplegia and aphasia prior to angiography. No vascular occlusion was demonstrated. A bad result.

RESULTS

The results following surgical treatment of 1 unruptured and 22 ruptured aneurysms are outlined in Table 1. The cases have been graded as to operative risk from 1 to 5. Grade 5 represents a moribund or near moribund patient with failing vital centres and extensor rigidity. Grade 4 includes patients with a major neurological deficit and deteriorating because of large intracerebral clots or older patients with less severe neurological deficit but pre-existing degenerative cerebrovascular disease. Grade 3 represents the drowsy patient with a neurological deficit and probably an intracerebral clot. Grade 2 indicates a drowsy patient without significant neurological deficit. Grade 1 indicates a patient who was conscious with or without signs of blood in the subarachnoid space.

The importance of assessing the results of both surgical and medical treatment in relation to the size and site of the aneurysm, age of the patient, time elapsed between rupture and operation and presence or absence of massive intracerebral or subdural clot has been emphasized by one of us (E.H.B.) in discussing surgery of aneurysms. Graf,[11] in an admirable paper, has recently pointed out the need for accumulating records of this type.

The results of surgical treatment in cases in which the patients survived have been graded. "Excellent result" means that the patient is well and has no neurological deficit. "Good result" indicates that there is a persisting neurological deficit, which is not disabling. The deficit may antedate or follow upon operation. "Fair result" indicates normal mentality and speech but disabling neurological deficit. "Bad result" indicates mentally retarded and dysphasic patients, and those with a major neurological deficit, e.g., hemiplegia or blindness would be classified as bad.

Walton's [23] studies of the mortality rate in subarachnoid haemorrhage

TABLE 1

Results following surgical treatment of aneurysms
22 Ruptured—3 deaths
1 Unruptured—1 death

Case No.	Age	Interval*	Coma	Semi-Coma	Clot	Op. Risk	Result
			INTERNAL CAROTID—SUPRACLINOID				
10†	41	4 hrs.	x		I.C.	5	Deceased
22†	26	10 hrs.		x	I.C.	2	Fair
9	36	25 days		Drowsy	I.C.	3	Excellent
13	36	36 days				1	Good
12	43	47 days				1	Good
			INTERNAL CAROTID—BIFURCATION				
14	16	4 days			S.D.	1	Excellent
2	34	44 days				1	Excellent
			ANTERIOR CEREBRAL: ANTERIOR COMMUNICATING				
20	50	2 days		x	I.C.	4	Deceased
3	36	4 days				1	Excellent
8	48	4 days		x		2	Excellent
11‡	49	7 days		x		4	Bad
1	41	11 days				1	Excellent
19†	42	16 days				1	Excellent
16†	41	24 days		Drowsy		2	Good
4	50	25 days				1	Excellent
17	52	39 days				1	Excellent
6†	47	44 days				1	Excellent
			MIDDLE CEREBRAL				
15	43	36 hrs.	x		I.C.	4	Excellent
18	58	35 hrs.	x		I.C.	4	Excellent
21†	50	5 days	x			4	Deceased
23	40	6 days	x			4	Bad
5	30	22 days				1	Good
7	58	Unruptured—Internal carotid				3	Deceased

* Between last haemorrhage and operation.
† Recurrent haemorrhage.
‡ Two aneurysms.
I.C. Intracerebral clot.
S.D. Subdural clot.

show that 15 per cent of the patients die in the first 24 hours, 12 per cent of the survivors by the end of the first week, 14 per cent of those survivors by the end of the second week, with a further reduction of 11 per cent at 4 weeks. Our experience indicates that the mortality rate in bleeding aneurysms diagnosed angiographically is higher than in the all-inclusive group labelled subarachnoid haemorrhage.

There were 12 cases in which operation was performed within 2 weeks; the result was excellent in 6, fair in 1, bad in 2 and 3 patients died. There were no deaths in the remaining 10 cases, with 6 excellent and 4 good results,

the patients having been operated upon between 14 days and 47 days after rupture. Among the surviving 19 patients postoperative angiograms were performed in 8 and in no instance was there evidence of an aneurysm.

An analysis of the 4 deaths* shows that in one instance hypothermia was directly responsible (Case 7), the only case of unruptured aneurysm (supraclinoid). Irreversible ventricular fibrillation was the result. The second patient (Case 10), with a supraclinoid aneurysm and massive intracerebral clot, was moribund. Extensor rigidity developed before operation and the brain stem haemorrhage found post mortem probably occurred at that stage. The third (Case 21), a diabetic hypertensive woman of 50 with a middle cerebral aneurysm, died of cerebral swelling, secondary to softening in the distribution of the middle cerebral artery, which probably antedated operation. A more generous decompression at surgery might have prevented this fatality. The fourth (Case 20), a known hypertensive aged 50, became hemiplegic following arteriography, which demonstrated an anterior cerebral-anterior communicating aneurysm. Paralysis of the left leg persisted after operation. The third postoperative day coma suddenly developed, with paralysis of the left arm and face, as well as the leg. She rapidly became decerebrate in spite of an emergency resection of her right frontal lobe, surviving at a vegetative level until her death 2 months later. Autopsy showed thrombosis of both anterior cerebral arteries.

HYPOTHERMIA

Increasing experience with the surgical treatment of ruptured aneurysms using hypothermia and, when necessary, bilateral cervical occlusion of carotid or carotid and vertebral arteries has increased our willingness to operate upon these patients promptly in the acute phase.

Brain Volume—Cerebral Swelling. At 30°C. (86°F.) and below, the brain usually has been "slack" unless there is a sizeable intracerebral clot or cerebral swelling secondary to intracerebral clot or cerebral softening. We have been misled on occasion by this decrease in brain volume. It was judged necessary to resect the frontal lobe only once in the course of operation in 10 cases of anterior cerebral-anterior communicating aneurysms. Re-exploration with frontal lobe resection became necessary in 2 of these cases, and in a third patient in whom the anterior communicating artery had been explored in addition to operating upon a middle cerebral aneurysm. It seems clear that cerebral swelling consequent to retraction of the brain, clipping of the parasagittal veins and manipulation of cerebral arteries is not reduced by hypothermia. The need remains for generous decompression and removal of traumatized brain. When the venous circulation to the frontal lobe is significantly impaired, frontal lobe resection is indicated.

Arterial Spasm. Marked arterial spasm has been observed in the course of manipulating the internal carotid in one case and in the middle cerebral artery in another case with the temperature at or below 30°C. (86°F.). The clinical course postoperatively has supported the diagnosis of arterial

* 3 deaths: ruptured aneurysm; 1 death: unruptured aneurysm.

spasm in these 2 cases and suggested spasm in 2 additional cases. It follows that hypothermia has not eliminated the problem of arterial spasm though a major degree of protection from anoxia is probably given to the brain during the period of hypothermia.

Reduction of Haemorrhage by Occlusion of Carotid or Carotid and Vertebral Arteries Bilaterally. In the course of operating upon 22 aneurysms which had bled previously, bilateral compression of the common carotid and vertebral arteries or common carotid arteries was judged by the surgeon to be very valuable in 11 cases. By this is meant that the surgical field obscured by blood in spite of efficient suction became relatively dry, that accurate definition and clipping or ligation of the neck of the aneurysm became possible and continuity of the artery was preserved.

In 4 cases (Table 2) gross reduction of bleeding followed interruption of carotid and vertebral arterial flow. Bilateral common carotid occlusion produced a relatively dry field in 5 instances when accompanied by systemic

TABLE 2

*Vascular occlusion**

Case No.	Age	Duration of Occlusion	Temp.	B.P. Mean	Reduction of Bleeding
		BILATERAL COMMON CAROTID			
1	41	7½ min.	30.7	58†	Dry
3	36	9¾+ 2¾ min.	28.4	64–36†	Relatively dry
6	47	13 min.	28.8	45†	Marked
12	43	4½ min.	30.8	80†	Moderate
16	41	11 min.	28.8	70†	Moderate
17	52	9+3 min.	30.0	100	Minimal
22	26	13½ min.	29.6	85	Minimal
		BILATERAL INTERNAL CAROTID			
8	48	6 min.	27.1	85	Marked
		BOTH COMMON CAROTID AND VERTEBRALS			
5	30	2 min.	30.2	90	Useful
9	36	11 min.	30.0	65 syst.†	Almost dry
11	49	6½ min.	29.4	60	Marked
19	42	8½ min.	30.2	90	Almost dry
		INNOMINATE PLUS COMMON CAROTID AND SUBCLAVIAN BELOW ORIGIN OF VERTEBRAL			
13	36	4½ min.	29.3	65	Dry
		VENTRICULAR FIBRILLATION			
4	50	14+55 min. cardiac massage	28.0	0	Dry
		RIGHT INTERNAL CAROTID			
10	41	10 min.	34.6	65 syst.	No effect

* Occlusion of carotid arteries by bulldog clamps and digital compression of vertebral arteries.
† Hexamethonium or Arfonad used.

hypotension. In 2 of 3 patients with normal blood pressure no significant decrease in bleeding followed bilateral common carotid artery occlusion, and in the third case there was a significant reduction. In 1 case bilateral internal carotid artery occlusion produced an almost dry field. Unilateral internal carotid artery compression proved ineffectual in 1 case.

Since the use of hypotensive drugs seems inadvisable at low temperatures bilateral internal or common carotid, or carotid and vertebral artery compression may be required in the management of anterior cerebral-anterior communicating aneurysms and in those cases of aneurysms of the internal carotid or middle cerebral artery in which bleeding may occur before exposure of the artery and aneurysm is complete.

The permissible period of occlusion of carotid and vertebral circulation using hypothermia will be discussed following the consideration of electroencephalographic studies.

ELECTROENCEPHALOGRAPHY

In 17 of the 23 cases under review, satisfactory pre- and postoperative studies and records during operation were obtained. Hypothermia had no demonstrable effect on the postoperative electroencephalogram. Changes observed following operation are comparable to the changes observed in similar operations without hypothermia. As cooling progresses theta activity becomes more pronounced. Below 30°C. (86°F.) the theta becomes more standardized with low voltage delta activity. Most patients at a temperature of 30°C. (86°F.) or below show no change in the electroencephalogram for 11 to 12 minutes after the cervical vessels are occluded bilaterally. A few patients show a slight increase in delta activity after 8 minutes. By contrast, in the conscious patient at normal body temperature, digital compression of one common carotid artery produces slow delta waves from the ipsilateral side in 10 to 12 seconds.[18] Similar observations have been reported by Aird.[24] The appearance of this delta activity has been used as a guide to the maximum time for which it is safe to occlude the vessels supplying the brain during operation.

SAFE PERIOD OF BILATERAL OCCLUSION OF CAROTID OR CAROTID AND VERTEBRAL ARTERIES

The part played by the basilar and each internal carotid artery in the formation of the circle of Willis, the blood pressure if the vertebral circulation is intact, and the age of the patient, are factors of major importance. Bilateral common carotid occlusion with bulldog clamps may reduce bleeding almost imperceptibly or produce a relatively dry field. In dogs Lougheed and Kahn[13] observed a 50 per cent reduction in cerebral metabolism at 30°C. (86°F.). Rosomoff and Holaday[17] reported 50 per cent reduction in cerebral oxygen consumption at 28°C. (82.4°F.). It follows that if the normal brain can withstand an interruption of cerebral circulation for 3 minutes at normal body temperature,[5,6] 6 minutes of cerebral ischemia at 30°C. (86°F.) should be well tolerated. Table 2 illustrates our experience with vascular occlusions. In only one instance (Case 4) has there been clinical evidence or

electroencephalographic evidence of local or general cerebral injury attributable to anoxia from interruption of the cerebral circulation. As recorded in the report of Case 4 this followed upon 14 minutes of ventricular fibrillation followed by 55 minutes of cardiac massage. Our purpose has been to minimize the duration of bilateral carotid and vertebral occlusion using intermittent arterial compression but the severity of bleeding and technical difficulties may dictate the course to be followed.

From our available information it appears that in humans the maximum period of common carotid or carotid and vertebral occlusion at 30°C. (86°F.) in a healthy individual of middle age or younger should not exceed 6 minutes and at 28°C. (82.4°F.) should not exceed 8 minutes. The period of safety is variable from case to case and the best determinant is the electroencephalogram which may dictate shortening or allow prolongation of the estimated safe period of occlusion.

ANAESTHESIA

Anaesthesia for neurosurgery with hypothermia should fulfill the following requirements:

(1) The anaesthetic agent should be eliminated by gaseous exchange without being metabolized. (2) The patient should be lightly anaesthetized and at the same time prevented from shivering with the development of anoxia or hypoxia.

In the first 10 cases Thiopental sodium was used and in retrospect we feel that this agent acting as a cardiorespiratory depressant contributed to the development of subclinical hypoxia, this, in turn, contributing to the high percentage of cardiac irregularities recorded in this group of patients. Chlorpromazine and promethazine have been used to potentiate the analgesic Demerol and nitrous oxide and trichlorethylene; a satisfactory anaesthesia has resulted meeting the requirements we have laid down. Dundee *et al.*[9] have shown chlorpromazine to be the most effective drug for the control of shivering. It hastens cooling perhaps by depression of the thermoregulatory centre. Additionally, being sympatholytic, it causes peripheral vasodilatation which, in turn, aids in heat loss. Subclinical shivering has been observed by the electroencephalogram and, if present, abolished by additional doses of intravenous chlorpromazine. Under these circumstances, significant respiratory depression has not occurred down to 28°C. (82.4°F.) and artificial respiration has not been necessary to improve the respiratory exchange.

CARDIOVASCULAR SYSTEM

Two cases of ventricular fibrillation in 33 coolings emphasize the importance of the cardiovascular system. Cardiac dysrhythmias are the commonest complications of hypothermia. Their prevention and control have taxed the minds of all workers in this field. Since 18 of our 23 patients had no known heart disease and no surgical interference to the heart was carried out, these patients have afforded an opportunity to determine the response of the normal heart to cold.[12] Hypothermia had no constant effect on heart

rate in this group of patients. One-third of the patients showed a slight increase in rate, one-third a decrease and the remainder no change. In each record there are temporary variations, lasting a few minutes, but these do not obscure the general trend. We have attributed this variation from the expected decrease, to the dysrhythmias encountered and the action of chlorpromazine.

The incidence and type of cardiac arrhythmia in 23 cases are given in Table 3. These results have been obtained by continuous electrocardiographic recording with one lead of the electroencephalograph, revealing ab-

TABLE 3

Incidence and type of cardiac arrhythmia

No. of Cases	Type of Arrhythmia	No. of Patients Who Received Pentothal
3	With heart disease known before operation	
	1 developed auricular fibrillation	
	1 continued auricular fibrillation	
	1 developed ventricular fibrillation	1
2	With pre-operative E.C.G. abnormalities	
	1 auricular flutter and fibrillation	
	1 auricular fibrillation	1
18	Without known heart disease	
	5 maintained sinus rhythm	1
	12 auricular arrhythmias	
	2 wandering pacemaker	
	1 auricular tachycardia	1
	1 auricular flutter	1
	8 auricular fibrillation	4
	1 ventricular fibrillation—preceded by auricular fibrillation	1
23		10

normalities that we have missed on the same electrocardiograph displayed on a cathode ray screen. Occlusion of the common carotid arteries often initiates a carotid sinus reflex, which produces a tachycardia. The cardiac rate is restored rapidly when the carotids are released. A significant drop in blood pressure occurred in 12 cases but this was expected in 8 cases because either a large intracerebral clot was evacuated or hypotensive drugs were administered.

Induced arterial hypotension by ganglionic-blocking drugs—hexamethonium bromide or Arfonad—has been employed in 7 cases. We feel that the use of these drugs may be hazardous because it was in 2 of the 7 cases that ventricular fibrillation occurred. This may have been chance occurrence in such a small number of cases but the fibrillation coincided with a rapid drop in blood pressure produced by the drugs. A sudden drop in blood pressure can produce an embarrassment of the coronary circulation and it seems logical to avoid as far as possible factors that might embarrass

the heart. Cookson has demonstrated that the quaternary nitrogens may have adverse effects on the myocardium during hypothermia (personal communication). However, the experience of Dundee *et al.*[8,10] supports the view that hypothermia affords protection against the effects of hypotension.

Analysis of the dysrhythmias listed in Table 3 shows that in those patients who received Pentothal there was a larger percentage of cardiac irregularities. The 2 patients with ventricular fibrillation received both Pentothal and Arfonad. These patients were aged 58 and 50 and auricular fibrillation developed as the rectal temperature approached 32°C. (89.6°F.) and ventricular fibrillation occurred at 28.6°C. (83.5°F.). Auricular fibrillation or flutter occurring above 32°C. (89.6°F.) we now regard as an indication to avoid temperatures below 30°C. (86°F.). Age itself is not a contraindication to hypothermia but it is well known that it predisposes to cardiac irregularities.

Temple Fay,[19] Talbot[22] and others demonstrated that auricular dysrhythmias were common occurrences in hypothermia, especially below 30°C. (86°F.), which disappear spontaneously upon rewarming. Ventricular fibrillation, obscure etiologically, is the one dysrhythmia that will terminate fatally unless treated promptly. Bigelow *et al.*[1,2] and also Swan *et al.*[20,21] felt that accumulation of carbon dioxide, which leads to a lowering of the pH and a shift of potassium may be instrumental in initiating ventricular fibrillation. They recommended hyperventilation to prevent acidosis and attempt to maintain the pH on the alkaline side. Churchill-Davidson[4] recommended positive negative respiration to improve the coronary blood flow.

We agree that these patients should not be allowed to become acidotic and if a pH change shifts to the acid side, then artificial respiration is necessary. However, so far respiratory excursion and volume have been adequate, and the pH has had a tendency to rise rather than fall.

SUMMARY

(1) Twenty-two patients with berry aneurysms have been operated on at varying intervals after rupture of the aneurysm, using hypothermia; there were 3 deaths. One patient with an unruptured aneurysm died after operation.

(2) Of 19 survivors 16 were classified as excellent or good, 1 fair, and 2 bad.

(3) Reduction of cerebral blood flow was achieved by bilateral occlusion of cervical vessels in various combinations. This procedure was of great value when the aneurysm was ruptured and allowed accurate identification and clipping or ligation of the aneurysmal neck.

(4) The electrocardiographic findings, the usefulness of the electroencephalogram, and the "safe" period of cervical arterial occlusion have been discussed.

CONCLUSION

It is our opinion that the results of surgery of intracranial aneurysms in this series of cases have been improved by the technique that has been re-

ported. We are encouraged to continue to use and to develop this method.

We are indebted to our colleagues Dr. K. G. McKenzie and Dr. T. P. Morley for allowing us to include their cases, to our medical colleagues for their support and collaboration in the management of these cases, to Miss Blackstock of the Department of Medical Art of the University of Toronto for the graphs, and to Miss E. Garrow and Mrs. A. Pyke for help in the preparation of the manuscript.

This study has been supported in part by a grant from the Atkinson Foundation and the Neurosurgical Research Fund of the University of Toronto.

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PERIPHERAL NERVE LENGTHENING

I. EXPERIMENTAL*

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(Received for publication August 22, 1955)

DURING the past 12 years, considerable advance has been made in the diagnosis and treatment of peripheral nerve lesions. However, the satisfactory repair of large gaps continues to be a problem. Grafts of all types have received extensive trials in humans and in experimental animals. Reports prior to 1940 have little value because of inadequate criteria for motor and sensory recovery. Furthermore, it has been difficult to apply to humans results obtained in animals because of different characteristics of regeneration. Heterogenous grafts have been universal failures. The encouraging results of homologous grafts in animals have not been equalled in humans. Autografts have been more successful.

In a well documented report, employing the Oxford scale for recovery, Seddon[7] reported that of 52 cases followed, 52 per cent showed results as good as suture, 67 per cent success or partial recovery, and 33 per cent were failures. There were 20 cases of digital, or other small nerve lesions with small gaps. Of main trunk grafts, the median was the most successful (7 success, 3 partial success, and 3 failures) while among various other main trunk grafts, 1 was a success, 1 partially successful, and 6 were failures. A serious disadvantage was that for defects in main nerve trunks, another injured nerve in the same patient had to be sacrificed for graft material, thus sacrificing one of the two nerves. In several instances in Seddon's series both nerves might have been salvaged by nerve lengthening.

Grafts present other disadvantages. As the length of a thick graft increases, there is increased danger of ischemic necrosis at the center of the graft, especially when the graft must be laid in a relatively avascular zone of scar secondary to the widespread area of tissue destruction so often associated with large nerve gaps. Finally, the growing axon, if it succeeds in traversing the graft, must then pass an additional suture line, which may be fibrosed by the time the axon reaches it.

Similarly, the procedures of extensive mobilization and transplantation

* This work was supported by The Naval Research Council: Contract N7ONR-401-1, Serial No. 5144.

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present certain disadvantages. Contiguous blood supply is interrupted, and postoperative scarring is engendered. When motion is restored and previously flexed joints are extended, traction may result in extensive damage over long segments proximal and distal to the suture line.[2,3] Extreme flexion of joints and immobilization are also highly undesirable in a denervated limb because of fibrosis, atrophy and contractures that occur.

On general grounds, therefore, the ideal method for overcoming large gaps is to transport by some means the central stump to the distal stump so that suture can be accomplished without tension and so that only the one suture line and distal stump need be traversed.

In a recent report, Hoen[4] noted the deleterious effects of traction on the distal segment of a severed nerve. It was also noted that when end-bulb suture was performed, the central end appeared to tolerate stretch better than the distal end. These observations led to the trial of several stretch procedures in patients presenting otherwise unbridgeable gaps; the results are the subject of a separate communication.[5] The present report deals with the initial phase of a program designed to evaluate these procedures in the experimental animal.

The early literature on nerve stretch has been summarized by Sanders[6] and Denny-Brown.[1] It is evident from a review of the literature that previous studies have been concerned with the effects of rapid or acute stretch. There has been no attempt to assay the effect of prolonged traction on nerves. The report of Denny-Brown cited above is of interest in that it points out that while a unifascicular nerve may elongate 100 per cent without serious damage, a multifascicular nerve will tolerate far less stretch without fatal damage, because of lesions of the integument, which is minimal in the unifascicular nerve. This suggests that the integument and not the axon is the limiting factor in nerve lengthening.

MATERIALS AND METHODS

Adult mongrel dogs weighing 15–20 lbs. were used. All were observed for a period of 1 month with serial weighing and limb measurement to rule out the possibility of growth before operation was undertaken.

The left sciatic nerve was used in all cases, the right serving as a control (except #5). The sciatic nerve, being a multifascicular nerve, was chosen because it was desired to test the effect of stretch on the integument as well as the axon. The anesthetic used was intravenous Nembutal and the nerve was approached posteriorly by separating the hamstring muscles. This allowed a clear approach to the nerve from the trochanter of the femur to the entry of the peroneal and tibial branches into the leg musculature. In the neuroma experiments, the nerve was sectioned, bleeding was controlled, and 6 weeks were allowed to elapse for the formation of a firm neuroma. Suture materials for attaching the neuroma included .007 and .003 tantalum wire and stainless steel wire #30 and #32. The steel wire proved best because it was less brittle than tantalum wire. The leg was immobilized by taping, plaster cast, or brace. The latter two were poorly tolerated by the animal and were discarded after a number of trials. The tape was removed in 2 weeks, and the animal was allowed to extend the leg spontaneously. This was usually carried out slowly over a period of

approximately a month, presumably being limited by pain.

Complications arising from denervation of the foot occasionally arose. The animal tended to walk on the dorsum of the denervated foot, with abrasions, swelling and infection, necessitating amputation in 2 cases in which the complication could not be controlled by bandaging.

The following types of procedures were carried out in order to find a satisfactory method:

A. Across the joint stretch of intact nerve.
B. Across the joint stretch of freshly cut nerve.
C. Across the joint stretch of nerve after neuroma formation.
D. Lengthening procedures using external traction wires.

At the termination of each experiment the animal was sacrificed by an overdose of Nembutal, and the lumbar cord, cauda equina, roots, sciatic, peroneal, and tibial nerves were removed in toto, placed on a cardboard mount without stretching, measured, and fixed in 10 per cent formalin. After fixation, the specimen was again measured and photographed. The specimen was then divided into numbered 2-cm. lengths, the numbers were recorded on the photograph, and longitudinal and cross sections were made of each 2-cm. segment.

The following stains were then done on spinal cord, roots, and the sciatic nerve and its branches: hematoxylin-eosin, Pal-Weigert or osmic acid for myelin, Gros-Bielschowsky for axis cylinders, and Laidlaw connective-tissue stain. Nissl stains were done on the spinal cord and dorsal roots for evaluation of changes in cell bodies. It was thus possible to compare operated and control nerve within any comparable 2-cm. section.

Some specimens show bubble formation in the myelin preparations. This is present in both control and operated sides and was caused by prolonged fixation in formalin necessitated by the fact that the preparations were made before staining facilities were available.

It will be noted in Tables 1–5 that 2 measurements are given: total and useful gains in length. The total length refers to the entire gross length of the nerve including neuroma. Reference points were: proximally, the level of the ischial tuberosity with the femur at right angles to the spine. Distally, the tibial nerves were always sectioned just at the medial malleolus so that the distal end of the tibia was a fixed point. With these two points known, the gross specimen could be accurately assembled so that the amount of lengthening, if any, could be determined. These measurements were checked at the time of autopsy and re-checked following fixation.

A microscopic study of the serially labelled sections was then made. The most distal point that was free of significant abnormalities and considered capable of supporting regeneration, as judged by the condition and size of the axons, myelin sheaths, and freedom from connective tissue, was determined, and the point identified on the working photograph. The distance from the ischial tuberosity to this point then determined the useful length, i.e., the length of nerve that appeared anatomically to be capable of supporting good regeneration. In all cases, myelinated fibers went far beyond this length but were reduced in numbers or were surrounded by excess connective

tissue, etc. Experience in our human series indicates that the criteria used in the animal series are much more rigid than is necessary, and that the figures given for useful gain have been underestimated. This assumption is based on regeneration studies following suture.

RESULTS

Controls. Two animals (Dogs 5 and 9) served as controls. The sciatic nerve was sectioned in mid-thigh, the ends being allowed to retract and lie

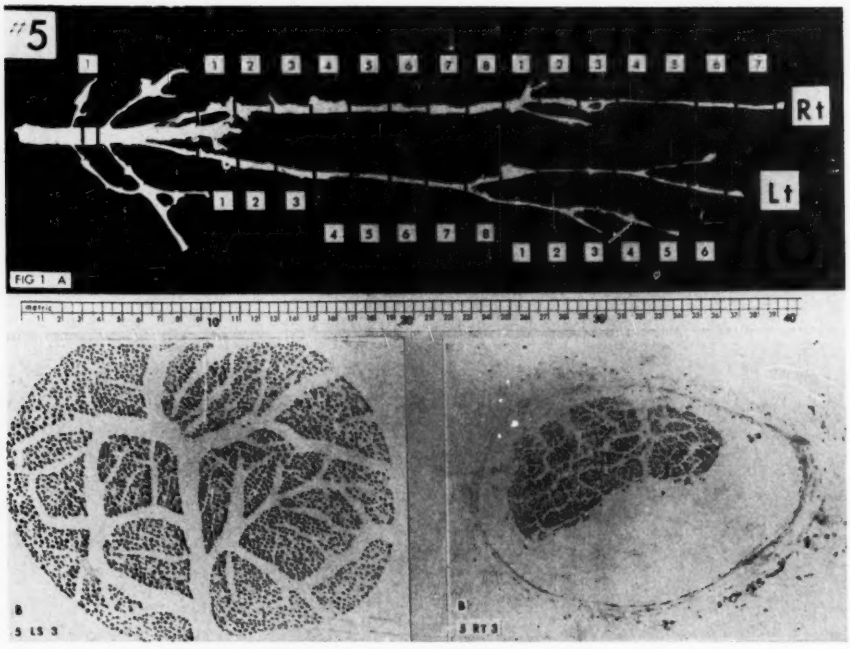

FIG. 1. *Control 5.* (A) Gross preparation to show site of nerve section (arrow) and fibrous bridge between proximal and distal segments. Rt.=right tibial; Lt.=left tibial. (B) Cross sections of left and right tibial nerves to show almost complete degeneration of the latter. Gros-Bielschowsky, ×220.

in the natural tissue planes. The animals were sacrificed at 15 months. Study showed that some axons had succeeded in penetrating the scar, and were present in the distal segment, but the distal segment was otherwise degenerated. Of interest was the variation in size and number of fibers in the proximal segment. These changes extended upward the entire length of the sciatic nerve (Fig. 1) and are probably related to the disunion of axon and peripheral ending, or possibly to persistence of some defects caused by retrograde wallerian degeneration. It may be postulated that axonal atrophy occurs when contact with end organ is lost. This appearance was similar to that reported by Weiss *et al.*[8]

Group A. Across the Joint Stretch of Intact Nerve. At the initial operation, the left sciatic was exposed from the ischial tuberosity to knee and, with the knee in acute flexion the nerve was carefully stretched to the point of resistance and then sutured to the fibula. The sutures were epineural as far as

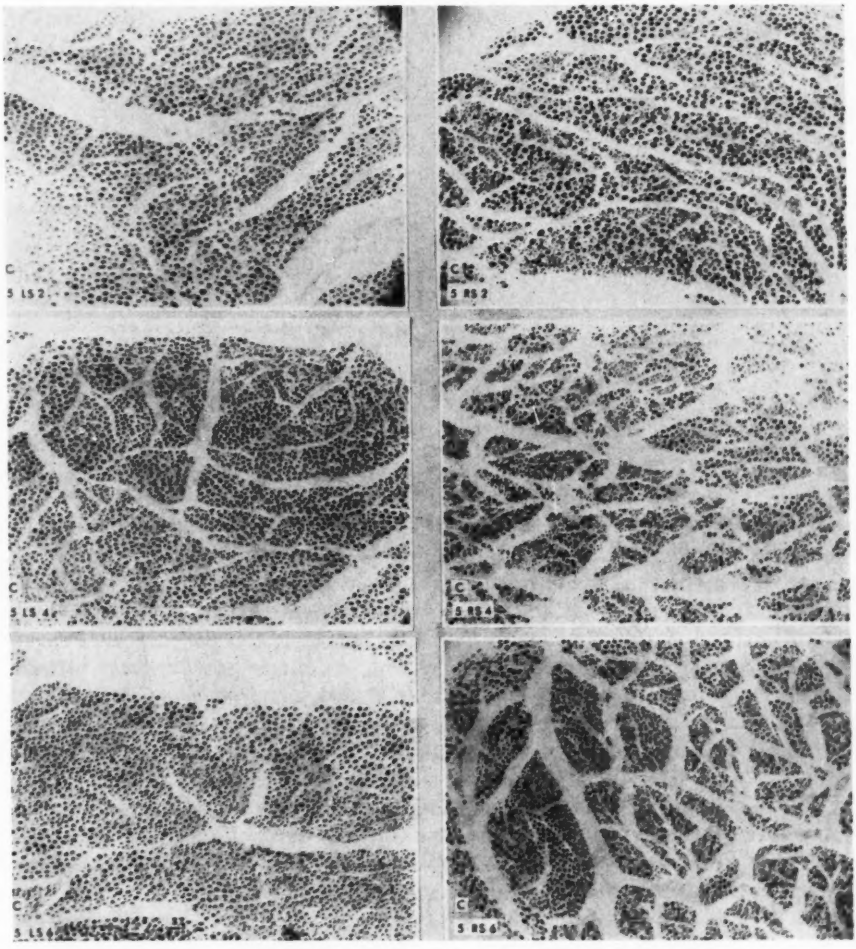

FIG. 1C. Cross sections from levels 2, 4, and 6 of sciatic nerves to show decrease in size and numbers of axons on right compared to control (left) side.

possible but inclusion of some nerve bundles did occur. After closure, the leg was taped in acute flexion for 1 week and then gradually extended over a 1-month period. The nerve was re-explored at monthly intervals to resuture the nerve at progressively distal points along the fibula. Two animals

TABLE 1

Results of various types of nerve lengthening procedures

Group and Dog No.	Type of Operation	Number of Operations	Length		Increase in Length			
			Initial Cm.	Final Cm.	Total Cm.	Total Per Cent	Useful Cm.	Useful Per Cent
A	Across joint stretch Intact nerve							
7		5	5	7	2	40	0	0
10		6	6	11	5	83	0	0
B	Across joint stretch Freshly cut nerve							
4		4	8	16	8	100	4	50
8		6	7	14	7	100	5	70
11		5	10	19	9	90	0	0
12	Tibial nerve	4	8	13	5	66	4	50
C	Across joint stretch After neuroma formation							
1		4	10.5	15	4.5	43	2.5	23
2		6	10	19	9.5	95	7	70
6		4	8	16	8	100	4	50

were used (Dogs 7 and 10).

Several features were common to both animals. It was impossible to maintain this type of traction suture in the nerve, the sutures pulling out at some stage of extension after each fixation. Hence, little if any lengthening resulted while the intact nerve was being stretched. After the third operative procedure, extraneural scar made movement of the joint and nerve difficult, and after the fifth operation it was no longer possible to recognize the nerve distal to the suture site. Table 2 indicates that not only did this method fail to produce useful lengthening of the nerve, but that severe retrograde changes also occurred high in the proximal sciatic nerve. The gross and microscopic appearance of such a preparation is shown in Fig. 2.

It was concluded that this method would not be satisfactory and the procedure was therefore abandoned.

Group B. Across the Joint Stretch of Freshly Cut Nerve. Four cases are included in this group: 3 sciatic nerves and 1 tibial (Table 3).

In Dog 4, after suture of the freshly cut proximal end to the fibula, the leg was taped in flexion 10 days and gradually extended. At re-exploration 3 weeks later, a 3.7-cm. increase in length had occurred. Epineural sutures were placed 5 cm. proximal to the end of the nerve, and stretch was repeated, similar procedures being carried out twice more. Despite wound disruption and healing by second intention, plus inevitable damage secondary to epineural suture as high as 7 cm. proximal to the nerve stump, intact axons were found to the neuroma. These were thinned over the distal

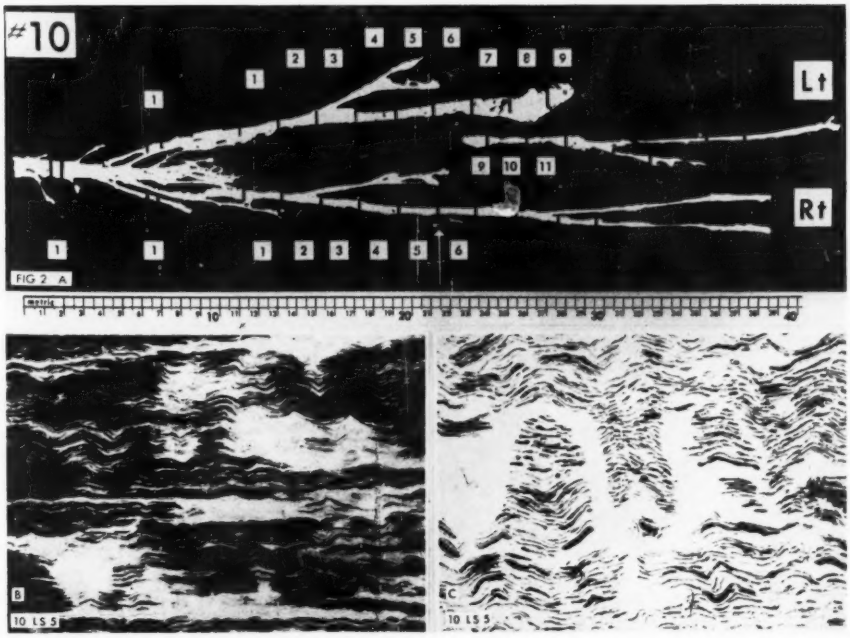

FIG. 2. *Dog. 10.* (A) Working photograph of gross specimen to illustrate method of numbering nerve sections. Rt and Lt = right and left tibial nerves. Note large neuroma, extraneural scarring, and diffuse fibrous enlargement of nerve. (B) Longitudinal section at level 5 of left sciatic nerve, showing patchy areas of necrosis and loss of myelin. Pal-Weigert, ×220. (C) Showing thinning and areas of loss of axons at same level as B. Gros-Bielschowsky, ×220.

8 cm., being markedly so over the last 5 cm., where there was heavy intra- and extraneural scar with thrombosis of the blood vessels and beading of the myelin, caused by the repeated operative procedures. An interesting feature of the Pal-Weigert preparations was increased distance between the nodes of Ranvier on the stretched side present over the last 8 cm. Another point of interest was that axons in intact muscle branches, leaving the stretched nerve central to the point of section and still connected to their end organs, were of normal caliber. This animal was sacrificed at the termination of the final stretch, no time being allowed for fiber maturation. Fig. 3 shows the gross and microscopic appearance of this preparation.

In Dog 8, the nerve was sutured at increasing distance down the fibula until it was necessary to resect 5.5 cm. of the nerve in order to carry out additional lengthening procedures. The resected portion was normal in all respects except for very slight thinning of the axons just proximal to the neuroma. The remaining nerve showed heavy extraneural scar, the result of 6 operative procedures, and intact axons with normal myelin down to the neuroma. The second neuroma was of course related to the final traction

TABLE 2

Cross joint stretch of intact nerve

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
7	9– 9–47	1. Suture left sciatic at point 5 cm. above knee to head of fibula in flexion	5		Some nerve bundles included in suture
	9–25–47	2. Nerve resutured to fibula head	5		Suture had pulled out of nerve
	11– 5–47	3. Nerve resutured to fibula 5 cm. below head	7		2 cm. increase in length
	1–28–48	4. Nerve resutured to fibula 5 cm. below head	7		Suture had pulled out of nerve. Moderate extraneural scar
	2–18–48	5. Nerve resutured to fibula 6 cm. below head	7		Very heavy extraneural scarring. No increase in length
	3–15–48	6. Sacrifice	7	0	Proximal neuroma formed at suture site and nerve could not be recognized distal to this. Nerve firm and hard for distal 3 cm. Marked extra- and intraneural scarring. No useful increase in length. Conclusion: A definite failure
10	10– 7–47	1. Suture left sciatic nerve at point 4 cm. above knee to fibula, 7 cm. below head	6		Brace used for controlled extension
	11–11–47	2. Resutured to fibula 6 cm. below head	6		Suture had pulled off fibula
	1–12–48	3. Resutured to fibula 1 cm. below head	6		Suture had pulled off fibula. Small amount extraneural scar
	1–29–48	4. Resutured to fibula 4 cm. below head	8		Suture had pulled off nerve. Very heavy extraneural scar
	2–16–48	5. Resutured to fibula 7 cm. below head	10		Nerve and suture intact. Nerve enlarged and edematous
	3–13–48	6. Resutured to fibula 7 cm. below head	10		Suture had pulled off fibula. Very heavy scar. Nerve enlarged, firm and edematous.
	7–20–48	7. Procedure terminated	11	0	Very dense extraneural scar and large neuroma; no recognizable nerve distal to this

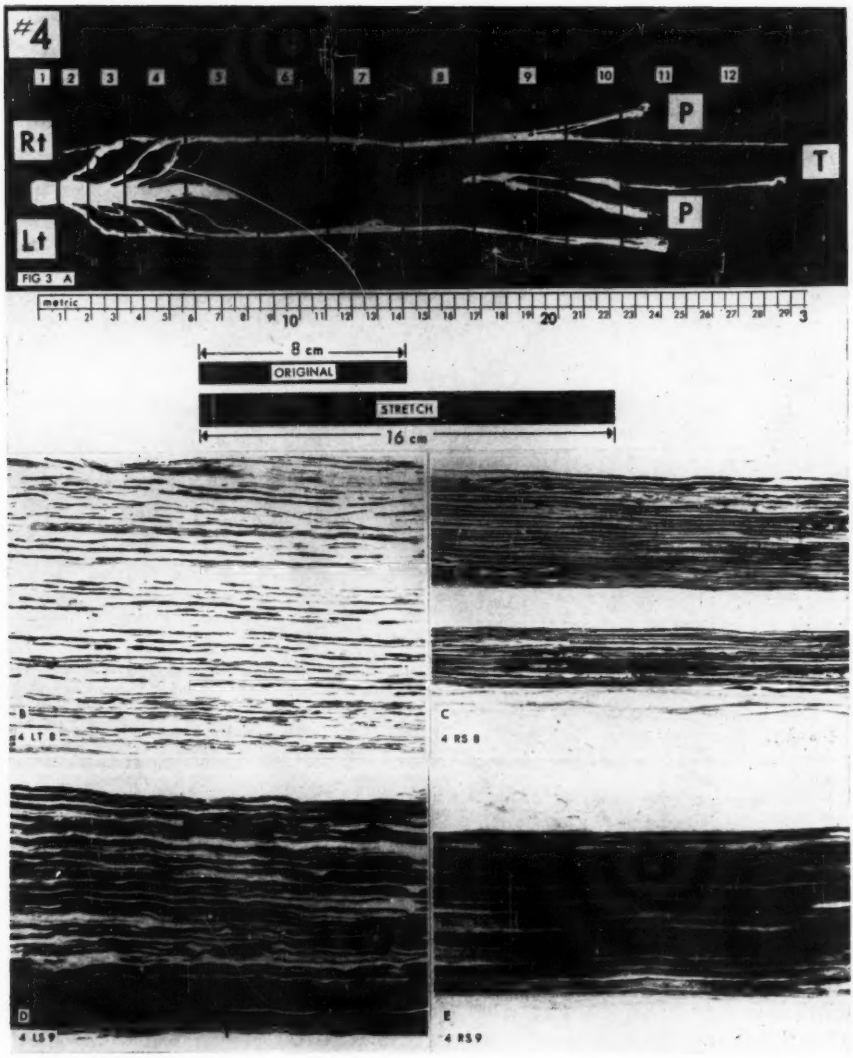

FIG. 3. *Dog. 4.* (A) Gross preparation to show total increase in length of nerve. Microscopic specimens taken at arrow. (B) Left and (C) right sciatic, showing intact but thinned axons in stretched portion of nerve. Gros-Bielschowsky, ×220. (D and E) Longitudinal sections of left and right sides respectively, to show myelin sheaths. Pal-Weigert, ×220.

AUTOPSY:

Gross: Entire nerve below sciatic notch enlarged 35 per cent and firm to palpation.

Micro: From sciatic notch downward the distal 14 cm. of nerve showed progressive thinning of axons, and reduction in size and number of heavy myelinated fibers. Marked intraneural edema and heavy intraneural scarring by proliferation of endoneural and perineural fibroblasts. Areas of ischemic scar are present. Epineurium is thickened. The distal 5 cm. is nearly all fibrous tissue. There was no increase in useful length and there were marked retrograde changes. Conclusion: A definite failure.

TABLE 3

Cross joint stretch of freshly cut nerve

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
4	8–13–47	1. Sciatic sectioned 2 cm. above knee and end secured to head of fibula in flexion	8		
	9– 4–47	2. Suture taken 5 cm.	11.7		3.7 cm. increase in length. Wound disruption postop.; healed by secondary intention
	10– 9–47	3. Suture taken 7 cm. proximal to end and attached to fibula	14		Additional 2 cm. lengthening. Scar tissue moderately heavy
	11–13–47	4. Suture taken 4 cm. proximal to end and attached to fibula 6 cm. below head	14		No additional lengthening
	12–16–47	5. Sacrifice	16	50%	Additional 2 cm. gained. Total gain of 8 cm. length

AUTOPSY

Gross: Distal 2 cm. thickened and firm to palpation. Average nerve diameters equal 2 sides.

Micro.: Axons present to base of neuroma, thinner than control; most marked at base of neuroma. Myelin shows widening between nodes of Ranvier and beading of myelin over final 3 cm. Very heavy extraneural scarring over distal 8 cm. with intraneural scar and thrombosed vessels over distal 5 cm. Useful increase in length was 4 cm.

sutures, and had the resection and resuture not been necessary, a 5-cm. increase in anatomically useful nerve would have occurred. This represents an increase of 70 per cent. Fig. 4 shows the gross and microscopic appearance of this prepration.

Dog 11 had 5 stretches over 126 days. At each operation the traction suture was found to be broken. Microscopic sections showed considerable damage to the nerve over the distal 15 cm., consisting of increase in cells, intraneural fibrosis, thinning and areas of disruption of axons and myelin sheaths. There were areas that strongly suggested the residuals of ischemic necrosis. We were unable to discover the reason for the too severe traction as evidenced by the wire breakage and deleterious results to the nerve.

In Dog 12 stretch of the posterior tibial nerve was carried out in 4 operations over 126 days. Sections showed thin but intact fibers with normal myelin to the base of the neuroma. These changes possibly resulted from compression by the extraneural scar, which was severe, plus lack of time for maturation of the fibers after the last stretch. It was estimated that a useful increase of 4 cm. had occurred.

Group C. Across the Joint Stretch after Neuroma Formation. In this group

TABLE 3—*Continued*

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
8	9– 9–47	1. Sciatic sectioned 5 cm. above knee and sutured to fibula in flexion	7		
	9–26–47	2. Suture taken at point 2 cm. proximal to neuroma and sutured to fibula	9		There had been a gain of 2 cm. in length
	10–21–47	3. Suture reapplied to fibula 4 cm. below fibula head	11		Additional 2 cm. gain in length
	11–20–47	4. Suture taken through tip of stump to fibula 9 cm. below fibula head in flexion	13		Additional 2 cm. in length; neuroma now well formed
	1–22–48	5. Distal 5.5 cm. nerve resected and fresh stump sutured to fibula 6 cm. below head	13		No further lengthening. Resected specimen was normal grossly with good fascicles.
	6– 1–48	6. No extension of leg noted			
	10–15–49	7. Sacrifice	8 + 5.5	12	Left sciatic was slightly thickened but otherwise normal in appearance. There had been a 1 cm. increase in length but distal 1.5 cm. was firm neuroma and scar

AUTOPSY

Gross: A. Resected portions grossly normal to neuroma. B. Distal 1.5 cm. wider than normal.

Micro.: Entire nerve shows slight thickening of epineurium and increase in perineural blood vessels. A. Resected portion: normal axons and myelin sheath and no increase in connective tissue to base of neuroma. B. Main nerve: aside from slight decrease in size of axons, and some increase in fibroblasts, this shows intact axons and normal myelin from neuroma to 2nd neuroma. Very heavy extraneural scar. Fibrous neuroma secondary to suture. Useful increase was 5 cm.

of 3 animals, the nerve was cleanly sectioned and 6 weeks were allowed for neuroma formation. This neuroma was then attached to the fibula and at monthly intervals the point of attachment was moved distally along the fibula. The results are summarized in Table 4. To this group should be added Dogs 8 and 12 from Group B (Table 3), who at some time in their course had had neuromas.

This group as a whole provided the most satisfactory method from a technical standpoint. The firm neuroma formed an ideal structure through which reliable traction sutures could be taken. There were only 3 instances of the traction wire pulling out. Injection of small amounts of alcohol into

TABLE 3—*Continued*

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
11	10–14–47	1. Sciatic nerve sectioned 5 cm. above knee and sutured to fibula 6 cm. below head in flexion	10		
	11–11–47	2. Nerve resutured to fibula	10		Suture had pulled out of nerve
	12–11–47	3. Nerve resutured to fibula 9 cm. below head	13		3 cm. increase in length; wire had pulled out of nerve
	1–23–48	4. Fresh proximal stump sutured to fibula, 4.5 cm. below head	15.5		Additional 2 cm. lengthening. Wire had pulled out. Distal 5.5 cm. resected of which 3.5 cm. appeared viable, although slightly firmer than normal
	2–20–48	5. Resutured to fibula 4.5 cm. below head	10		2 cm. increase in length. Moderate scarring extraneurally and distal 2 cm. of nerve very firm. Wire sutures had broken
		6. Sacrifice	14	None	2 cm. additional increase in length. Very heavy extraneural scar. Distal 3 cm. nerve very firm. Total nerve lengthening was 8 cm.

AUTOPSY

Gross: Entire nerve including resected portion was slightly thickened and firm. This was most marked over distal 7 cm. of 2nd stretch.

Micro.: A. Resected 5.5 cm.: very heavy increase in fibroblasts and perineural connective tissue. Axons very thin; myelin disrupted. B. Main nerve: distal 15 cm. shows marked increase in fibroblasts, proliferation of all connective-tissue elements, very thin axons greatly reduced in number, and poor myelin distally. Frequent areas of disruption possibly secondary to ischemic necrosis. Useful increase: none, a definite failure.

the neuroma rendered it non-painful and stretch was well tolerated. The neuroma provided a broad cross-section surface so that traction was evenly distributed to integument and nerve structure. Table 4 shows that in Dogs 1 and 6 substantial increases in useful length were yielded in 3 operations, contrasted with a greater number of procedures for other types of lengthening.

In Dog 1, despite heavy extraneural scarring, there was an increase of 2.5 cm. of useful nerve.

A total of 8-cm. lengthening had been achieved in Dog 6 when the experiment was interrupted by the accidental death of the animal. The distal 4 cm. exhibited extra- and intraneural scarring secondary to suture pro-

TABLE 3—*Continued*

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
12	10–28–47	1. Posterior tibial nerve sectioned 4 cm. above knee and sutured to fibula 6 cm. below head in flexion	Ischial tuberosity to cut end of tibial nerve —8 cm.		
	12–28–47	2. Resutured to fibula	8		Suture had pulled off fibula
	1–26–48	3. Resutured to fibula 7 cm. below head	9		Considerable extraneural scarring
	2–24–48	4. Resutured to fibula 10 cm. below head	13		Additional 4 cm. increase in length. Heavy scarring and nerve narrowed 4 cm. proximal to end
	3– 8–48	5. Found dead; autopsy done	13	12	There had been an increase of 5 cm. in length of tibial nerve. Diameters were: rt. tib. 2.5 mm.; lt. tib. 2.0 mm. Neuroma was 1.5 cm. in length; remaining nerve felt firmer than normal. There was heavy extraneural scarring

AUTOPSY

Gross: Entire tibial nerve was firmer than normal and slightly thinned.

Micro.: Heavy extraneural scar, but no increase of connective tissue within the nerve. The axons are thin, but intact to the neuroma. The myelin is normal. Useful increase in length was 4 cm.

cedures, but there was an additional increase of 4 cm. of essentially normal nerve (Fig. 5).

Dog 2, the most promising grossly of the entire series, cannot be reported in detail because of the accidental fixation of the specimen in high-strength formaldehyde, with consequent ruination of the specimen. This animal, with an initial length of 10 cm., showed an increase in length of 9.5 cm. before removal of the specimen was necessitated by the fact that the neuroma was at the ankle and a more distal attachment was not available. Section at the base of the neuroma showed a completely normal nerve grossly. The nerve was normal to palpation and of normal size. Bleeding was excellent from the cut end and the integument appeared normal. The fascicles of both peroneal and tibial divisions appeared completely normal. It was estimated that at least 7 cm. of this increased length was normal nerve suitable for suture. The details of Dogs 8 and 12 are found under Group B.

Group D. Lengthening Procedures Using Traction Wires. In certain cases encountered clinically, the proximal segment of the severed nerve was so

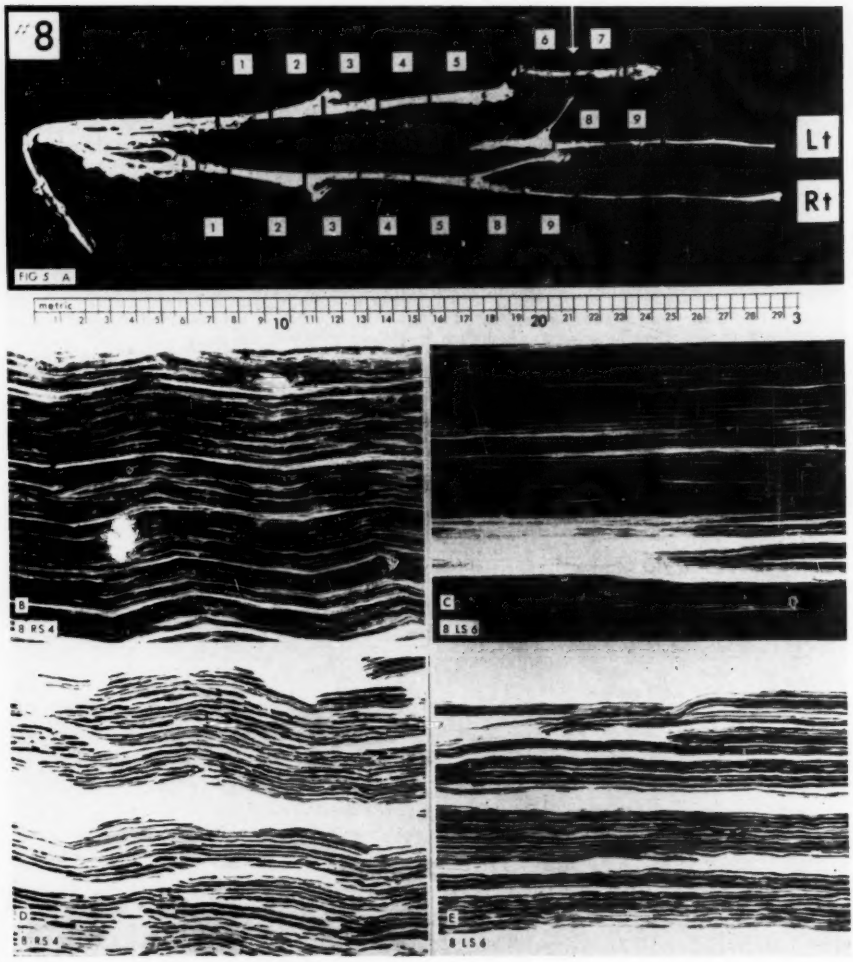

FIG. 4. *Dog. 8.* (A) Gross specimen. Note normal appearance of resected portion. Neuroma of main nerve secondary to resuturing. Microscopic specimens taken at arrow. (B) Right control and (C) left stretched sciatic to show normal myelin. Pal-Weigert, ×220. (D) Right control and (E) left sciatic to show normal appearance of axons on the stretched side. Gros-Bielschowsky, ×220.

short that cross-joint stretch could not be done. To meet this difficulty, traction wire and vitallium traction heads have been devised and used with success.[5] It was desired to attempt to evaluate these procedures in the animal. In 6 animals, high sciatic section was carried out and a vitallium head was attached, with traction wire attached to the fibula with the leg in flexion. In 4 of these cases, a sleeve of medically pure polyethylene sheet was placed about the nerve and wire to facilitate movement of the traction

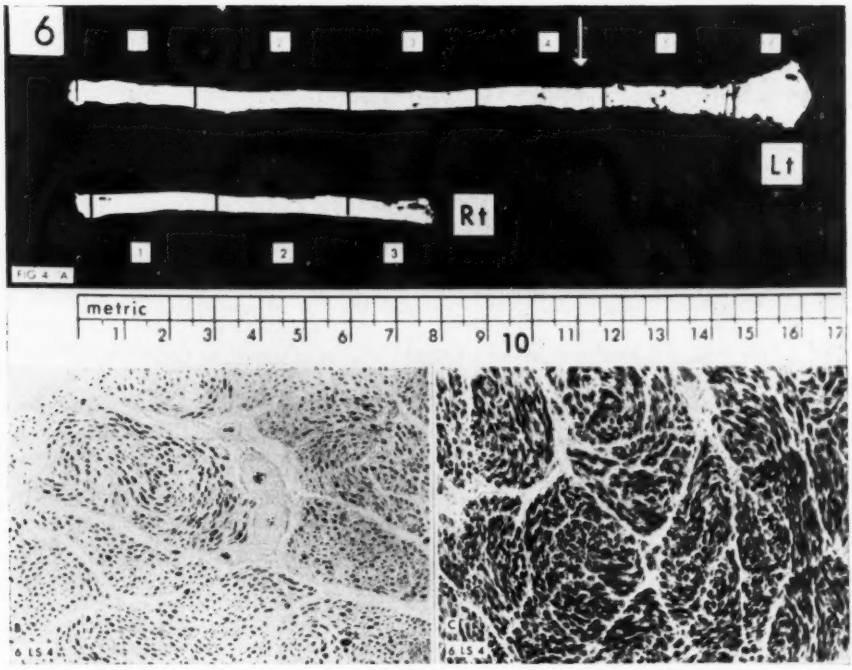

FIG. 5. *Dog. 6.* (A) Gross preparation. Both nerves were sectioned at ischial tuberosity for comparison. Microscopic specimens taken at arrow. (B) Showing normal axons. Gros-Bielschowsky, ×220. (C) Same area as in B. Pal-Weigert, ×220.

head and nerve. In each case, adhesions were formed about the nerve and between the nerve and the film. In addition, a thin serous fluid was always produced. The film tended to fragment in 2 months. Fibrin film was tried in 2 cases and was well tolerated but fragmented early. In each case in which fluid was formed, it ultimately became infected, invalidating the experiment. It was not possible to achieve any satisfactory results with this type of procedure, within the time limits of the investigation. In humans, external traction procedures had been well tolerated.

An effort was made to devise a satisfactory type of external traction in the dog. After neuroma formation, the traction head was attached to the nerve, and the wire was brought out through a sealed stab wound to a turnbuckle attached to a cast. Each day the slack on the wire was carefully taken up. In another type of procedure, the cut nerve was reflected to the subcutaneous tissues of the back and traction was carried out as before, utilizing a vertebral spine as a traction point. Some of these procedures were carried out without sleeves, while in others the nerves were incorporated in medically pure polyethylene sleeves. In the former group all lesions became infected about the second week, the infection passing in along the traction

TABLE 4

Across the joint stretch after neuroma formation

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
1	8–13–47	1. Sciatic sectioned 10.5 cm. below ischial tuberosity	10.5		
	9–10–47	2. Neuroma sutured to neck of fibula. Cast in full flexion	10.5		
	9–30–47	3. Resutured to fibula below neck	13.0		Increase of 2.5 cm. of normal-appearing nerve
	10–28–47	4. Resutured to fibula 7 cm. below neck	?		Heavy extraneural scarring. Degree of lengthening not noted
	12– 2–47	5. Sacrifice	15.0	13	Heavy extraneural scar

AUTOPSY

Gross: Both nerves measured 8 mm. in diameter. Moderate terminal neuroma at suture site, otherwise normal to inspection and palpation. Over-all increase of 4.5 cm.

Micro.: Axons present through neuroma; thin over distal 4 cm. with faint myelin. Very heavy extraneural scar; no intraneural fibrosis. Useful increase was 2.5 cm.

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
2	8–13–47	1. Sciatic sectioned 10 cm. below tuberosity	10		
	9–12–47	2. Neuroma sutured to mid fibula	10		
	9–30–47	3. Resutured to fibula	12		Nerve had come loose but 2 cm. increase in length
	12–23–47	4. Resutured to fibula			Suture broken
	2– 4–48	5. Resutured to fibula 9 cm. below head	16		Suture broken but marked increase in length, appearing normal
	2–19–48	6. Resutured to malleolus 12 cm. below head of fibula	19		Intact
	3–10–48	7. Most distal 10 cm. resected for specimen	19.5	17.0	Nerve immobilized by marked scar

AUTOPSY

Gross: Left sciatic, including resected portion, entirely normal. Both tibial and peroneal division measured 2 mm. in diameter, equaling those of the right. The fascicles were entirely normal proximally and in a section made at base of neuroma. Estimated useful increase in length was 7 cm. (See text)

Micro.: None. By error this specimen was fixed in 25 per cent formalin with marked charring and shrinkage of tissue.

TABLE 4—*Continued*

Dog No.	Date	Operation	Length		Result
			Total Cm.	Useful Cm.	
6	9– 8–47	1. Section left sciatic 8 cm. below ischial tuberosity	8		
	10– 2–47	2. Good neuroma. Sutured to fibula 9 cm. below head	8		In traction 4 days; badly tolerated
	11– 6–47	3. Suture 3 cm. proximal to neuroma attached 10 cm. below knee	12		Nerve appeared normal
	2– 6–48	4. Resuture of neuroma to malleolus	15		Nerve slightly joined over distal 3 cm.
	3– 8–48	5. Found dead. Control right sciatic 10 cm. long	16	12	Increase of 8 cm. including 1.5 cm. neuroma; 2.5 cm. segment proximal to neuroma firmer than normal and slightly thicker. Diameters: rt. 3 mm.; lt. 4 mm.

AUTOPSY

Gross: Epineurium thick and rough over distal 3 cm. Nerve firm to palpation; otherwise normal.

Micro.: Epineurium thickened over distal 10 cm.; otherwise normal to distal 3 cm. when there is marked intra- and extraneural fibrosis, thinning of fibers and fragmentation of myelin. This was caused by sutures taken in operation 3. Useful increase was 4 cm.

wire, while in all of the latter group fluid developed on the third to sixth day which then became infected. Several types of medically pure polyethylene sheeting were used, as recommended in previous publications, all without success despite special efforts to prevent infection.

Thus, these methods, already successfully used in the human, could not be adapted to the experimental animal within the scope of the present project. All of the external traction procedures tried in 21 dogs were failures because of fluid formation or infection.

DISCUSSION

Some considerations regarding the problem of closure of large gaps, with particular reference to nerve grafts, have been presented. Although autogenous grafts have been successful, donor nerves of suitable size may not be available, and cable grafts have been disappointing. It was because of these considerations that nerve lengthening was undertaken as a last resort in a group of clinical cases of otherwise unbridgeable gaps. Initial success and satisfactory motor and sensory recovery despite severe nerve and soft-tissue lesions led to the continuation and improvement of the procedure. The results of these experiences are recorded by Hoen.[5]

This first laboratory effort was directed toward developing a satisfactory experimental method of nerve elongation, gaining some idea of the amount

of lengthening that might be achieved, and the anatomic changes that resulted therefrom.

To date, the most satisfactory type of lengthening procedure has been across the joint stretch after neuroma formation. The advantages of this type of procedure have been listed and technically it is by far the best. With lengthening taking place across the joint slowly, the tissues tend to move with the nerve so that suture breakage and pull-outs are very infrequent. Substantial increases in length may be obtained in 3 operations and possibly less. Intact and freshly cut nerve stretches have not been successful.

A criticism of the method is that it does not allow for satisfactory control of extension of the limb. The use of braces and extensible casts was not satisfactory and while uncontrolled extension by the animal appeared to be fairly constant and gradual, one could not be certain that the limb had not been subject to sudden strains or increases in extension which might produce deleterious effects on the nerve. It was to obviate these difficulties that a number of types of external traction procedures were explored.

External traction procedures were not successful because of infection along the traction wire, and the use of buried plastic sleeves was also unsuccessful because of fluid formation and subsequent infection. It is clear, however, that further efforts along this line are indicated since this method utilizing the external traction wire offers the best possibility of precise control of nerve elongation, especially in respect to time.

Despite the exploratory nature of this initial experience, it was possible to achieve increases in length of 2.5 to 9.5 cm. in the dog's sciatic which normally measures less than 10 cm. after emergence from the pelvis. Six of 9 animals showed anatomically useful increases of 2.5 to 5 cm., in which there were no changes from normal structure, save some decrease in diameter of axons, similar to that seen in the controls when the nerve was simply severed. It is possible that even greater increases may be obtained with few operations and neuroma suture only.

A number of anatomic changes were found in each case after stretch, particularly in those cases considered failures.

Grossly, the nerves appeared normal to palpation and inspection except distally in the region of the suture where the nerve was thickened and firm. The surface vessels appeared normal. Diameters of stretched and control nerves were the same. On fresh section, the fascicles appeared normal, protruded in a normal fashion, and the cut ends bled normally. The nerve appeared to have a normal amount of elasticity.

Microscopically, the epineurium was only slightly thickened except distally as the region of repeated operative procedures and suture was reached. Here a gradually increased thickening of the epineurium was noted as it then blended into extraneural scar secondary to the operative procedures. The vasculature on the surface of the nerves was grossly normal; detailed injection studies of the circulation have not been carried out. Neurolemmal hernias and pseudoneuromas as reported by Denny-Brown

and Doherty[1] in acute nerve stretches were not encountered. Areas of ischemic necrosis were not seen except where mentioned.

Intraneurally, the perineurium and endoneurium were thin and delicate in all cases except as specifically noted. Thickened intraneural connective tissue was of course present beneath the operative areas. At autopsy, premortem bleeding from the intraneural vessels appeared normal.

In all cases, there was decrease in diameter of the axons throughout the central stump up to the level of the lumbosacral plexus. Two points were of interest: similar changes were found throughout the central stump of the control animals (Fig. 1) and such changes were not present in those upper intact branches innervating the hamstring muscles. It seems likely that this thinning of the axons was the result of one or more of three causes: (i) retrograde wallerian degeneration secondary to section and repeated trauma; (ii) prolonged disunion with peripheral end organs; or (iii) actual stretching of the axon.

It has been shown[8] that when a central stump is prevented from regenerating to the distal end organ, a decrease in the number and diameter of the fibers of the central stump occurs. This was certainly the case in the present series, in that while being extended the central segment was never in contact with the distal segment, and hence was deprived of connection with affector mechanism. These changes were the same in the control and stretch animals. An exception in both groups were those fibers that innervated branches to the hamstring muscles. These fibers, connected to their end organs, appeared to be unreduced in number and of full diameters. Hence there is reason to suspect that the decreases in diameter of the fibers found in these stretched preparations are partly, if not largely, attributable to the fact that the central stump was disconnected from the end organ over long periods of time. The relative role of this factor and possible elongation of the axon by stretch in producing decrease in diameter of the fiber will be the subject for further study, and will require regeneration experiments for its resolution.

In many cases it appeared that massive extraneural scar secondary to repeated operative procedures may have produced thinning of the nerve fibers (particularly the heavily myelinated fibers), since these effects became more noticeable in relation to heavy scarring. A striking feature, however, was the unusual freedom from intraneural fibrosis as demonstrated by the Laidlaw connective-tissue stain.

A survey of the anterior horns and dorsal root ganglia failed to reveal significant difference in number or appearance of nerve cells. Cell population counts were not made however.

An entirely different picture was seen in the 3 cases (Dogs 7, 10 and 11) in which the operation failed to produce useful lengthening of the nerve. Here there were heavy increase in cellular elements, increase in endoneural fibrous tissue, areas of ischemic necrosis, and marked loss of myelin and nerve fibers.

From evidence thus far available, it appears that the limiting factor in nerve elongation is not the axon but the integument. Initial experience with slow elongation suggests that suitable methods for a significant nerve lengthening without damage may be developed.

SUMMARY

The results of an attempt to produce elongation of a multifascicular nerve are reported. Methods investigated and anatomical results achieved are described. While definitive results await regeneration studies following nerve elongation, the present study indicates that significant increase in nerve length may be obtained.

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ISCHEMIA OF THE SPINAL CORD

AN EXPERIMENTAL STUDY*

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(Received for publication September 6, 1955)

CAVITATION of the spinal cord has been reported in association with many different disease processes. There is increasing evidence that many of these secondary cavitations are produced by spinal cord ischemia. While the etiological sequences leading to spontaneous syringomyelia remain more obscure, some evidence has accumulated that they too involve vascular factors.

As early as 1887 Joffroy and Achard[3] reported spinal cord cavitations in cases of hypertrophic pachymeningitis which they felt were caused by venous stasis and arterial thrombosis. Camus and Roussy[1] made a similar report in 1914. More recently Lubin[5] described spinal cord cavitation in cases of adhesive spinal arachnoiditis. Lichtenstein[4] ascribed cavitations associated with Arnold-Chiari malformations to compression of the vertebral arteries and venous stasis.

Experimental work supports the vascular etiology of some cavities. Tauber and Langworthy[8] produced spinal cord cavitation in cats by ligation of the anterior spinal artery. Several authors have produced spinal cord cavitations secondary to experimental arachnoiditis. Such cavitations were studied by McLaurin *et al.*[6] who agreed with previous investigators that the lesions result from spinal cord ischemia. Their paper includes an excellent review of the literature on this subject.

The present investigation is intended to establish more clearly the nature of lesions produced by uncomplicated spinal cord ischemia. Special attention was given to avoid changes caused by surgery and other non-vascular factors.

MATERIALS AND METHODS

In all experiments, laminectomy was conducted under strict aseptic conditions in the mid-thoracic spine under pentobarbital sodium anesthesia. Healthy adult mongrel dogs weighing from 8.0 to 16.0 kg. were used.

Preliminary experiments showed that interruption of the superficial

* This work was supported by research grant B-80(c) from the National Institute of Neurological Diseases and Blindness of the National Institutes of Health, Public Health Service, Department of Health, Education, and Welfare.

vessels of the spinal cord either by tearing, ligation, or coagulation produced so much ancillary surgical damage that clear-cut ischemic changes could not be delineated. Likewise, ligation of the anterior spinal artery alone or combined with multiple intradural root section—although resulting in demonstrable neurological deficits—resulted in a considerable amount of undesired scarring. Only when the dura mater was not opened were the histological changes secondary to careful surgery found to be minimal. Efforts were made to isolate the radicular blood vessels in the nerve root sleeves but such surgery again produced considerable scarring. It was therefore concluded that the radicular blood supply to the mid-thoracic spinal cord could best be

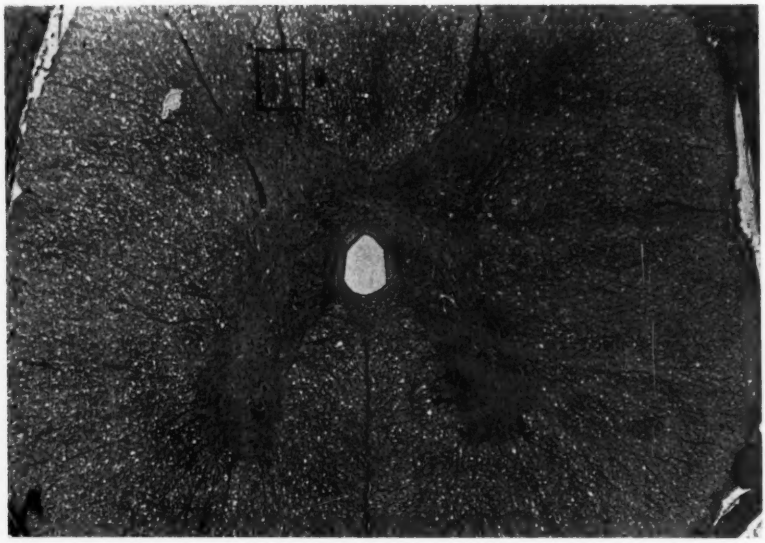

FIG. 1A. Cross section of spinal cord at T6 (hematoxylin and eosin ×38) from animal subjected to extradural radiculectomy of T5, T6, T7, T8, T9, and T10. Note especially the loss of dorsal horn cells and the generalized gliosis of the gray matter.

interrupted by sectioning of the entire nerve root along with the blood vessels. Animals prepared by the section of from 1 to 4 sets of adjacent nerve roots showed either no neurological deficits or such deficits were extremely transient. However, this material served as good control for the changes induced by nerve section alone. Animals with 5 sets of adjacent nerve roots sectioned frequently showed transient neurological deficits. When the roots from 6 adjacent spinal cord segments were sectioned there was temporary paraplegia which on occasion proved to be replaced by paraparesis. This level was then taken as the basis for the present study.

A total of 42 dogs were prepared by the section of 6 adjacent pairs of spinal roots extradurally. These pairs usually included T6 through T11. Of these, 27 sur-

vived adequately for good postmortem studies including removal and examination of the spinal cord. These animals were sacrificed between 1 to 4 weeks after the initial surgery. From these the spinal cords of 16 were selected for careful histological studies. Each of these spinal cords was studied at many levels above and below the laminectomy as well as at the point of surgical attack. After proper fixation of the entire central nervous system, tissues were prepared in the routine manner and were sectioned and stained by various methods. Hematoxylin-eosin was used routinely, and in selected sections the gold chloride sublimate method for astrocytes and the Gros-Schultze method for axis cylinders were used. Occasional sections were stained with Sudan Black.

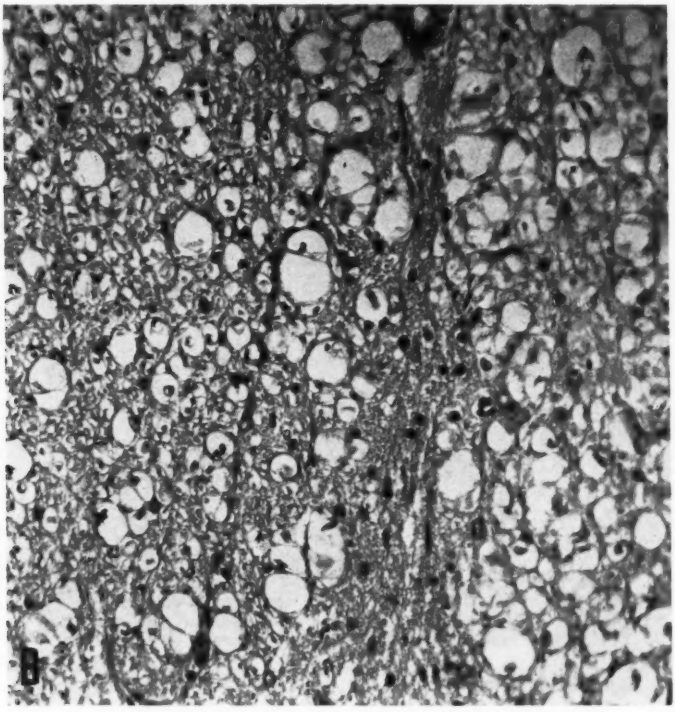

FIG. 1B. Area outlined in Fig. 1A magnified ×300 to demonstrate more clearly the loss of dorsal horn cells.

RESULTS

Clinically, the animals with ligation and section of 6 adjacent thoracic nerve root pairs with the accompanying blood vessels showed a band of loss of sensation corresponding to the nerve roots sectioned. In addition there was a caudad sensory loss of pain sensation which often reached up as far as the thoracic segments, especially in those animals showing paraplegia. In animals showing only paraparesis and inco-ordination there was a sensory

sparing in the caudal area which usually was present up to the lower lumbar dermatomes. Most of the animals showed these changes transiently, with the evidences of long-tract impairment being gone in 1 to 2 weeks.

Histologically, the most consistent and widespread change in all cases receiving microscopic examination was the loss of dorsal horn neurons, those of Clark's column being affected most severely. Between the area of neuron loss and the less disturbed anterior horns there often was found a zone of

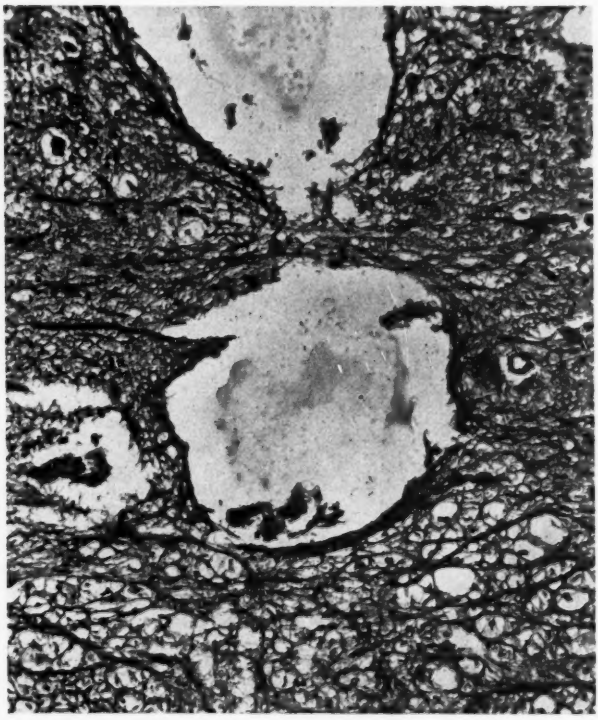

FIG. 2. Cross section of spinal cord at T9 (hematoxylin and eosin ×155). Section taken 10μ from point where central canal with otherwise intact ependymal lining communicates with cavity in the dorsal columns beyond the top of this microphotograph.

incomplete neuron damage characterized by fragmentation, satellitosis, and neuronophagia. The coagulation and sclerotic type of nerve cell change was observed less frequently. This pattern of nerve cell damage seemed to be affected very little by differences in chronicity of the lesions. It is of particular interest that in the few experiments that failed to produce cavitation, the only clear-cut pathological change was the loss of dorsal horn neurons (Figs. 1 A and B). In most experiments the neuron loss extended several spinal cord segments above and below the region of experimentation.

Much less definite than the neuron damage, but coexistent with it, was

a mild generalized gliosis which involved the gray matter diffusely. This was seen best in the gold chloride preparations.

Cavitation was seen in 12 of the 16 spinal cords studied histologically. Most of these cavities were small, only one being noted grossly. The midline gray matter dorsal to the central canal was always involved at some level of each cavity. The larger cavities sometimes extended from this point along the dorsal horns, often involving the adjacent white matter of the posterior or lateral funiculi. Communication of the cavity with the central

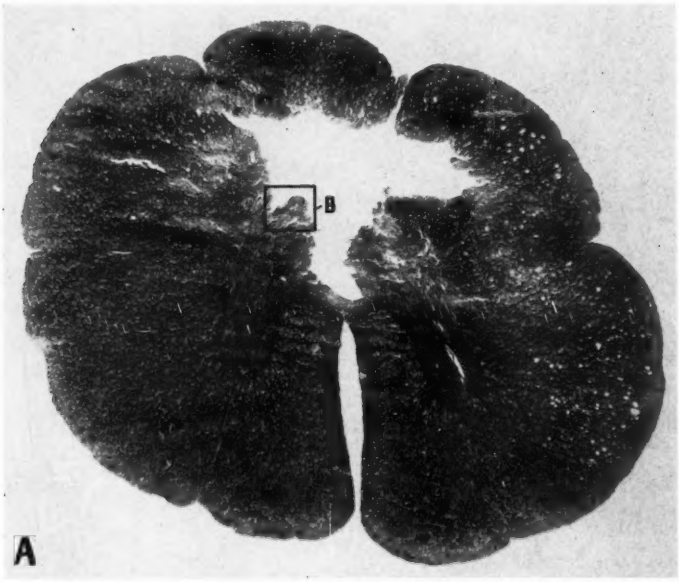

FIG. 3A. Cross section of spinal cord at T8 (gold sublimate ×25) showing extensive cavitation from the gray matter dorsal to the central canal extending posteriorly over much of the dorsal funiculi through one dorsal horn and well into the lateral funiculus of that side. There is generalized gliosis with hypertrophy of the vascular feet.

canal was a frequent finding (Fig. 2), but no attempt was made to demonstrate this in all cases.

Reactive changes in the interstitial neural elements were of mild degree and late development. Hematoxylin-eosin sections failed to show definite glial hyperplasia in the vicinity of the cavitation. Gold chloride preparations in some of the more chronic cavities demonstrated a characteristic change in the astrocytes bordering the cavity. These astrocytes had few processes but they were greatly hypertrophied and extended toward and into the cavity. In some places, the wall of the cavity appeared to be a palisade of these hypertrophied processes (Figs. 3 A and B). Gitter cell response occurred when the cavity involved the white matter with actual destruction

of the traversing nerve fibers (Figs. 4 A and B). In some cases the cavity extended medially and posteriorly for a short distance into the dorsal funiculus without evidence of nerve fiber destruction. In such instances, no significant phagocytosis was present (Fig. 1).

The central canal of some specimens was greatly enlarged and definitely abnormal in the region of ischemia. In most cases, however, the enlargement

FIG. 3B. Area outlined in Fig. 3A magnified ×300, demonstrating the tendency to palisading of the hypertrophied fibers of the astrocytes bordering the cavity.

was only slight and could not be considered pathological without further study.

By comparison, the experiments involving intradural surgery showed that the leptomeninges were markedly fibrotic and often were incorporated in the dural scar. When the ischemia was produced by extradural means, the leptomeninges showed only minimal fibrosis, and adhesions were absent. In the latter experiments, considerable condensation of connective tissue

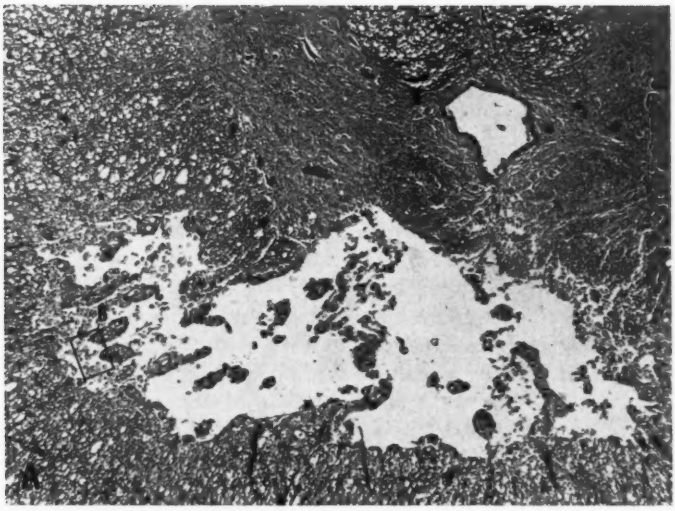

FIG. 4A. Cross section of spinal cord at T8 (hematoxylin and eosin ×50) showing cavitation in the white matter in which there is active phagocytosis with many gitter cells.

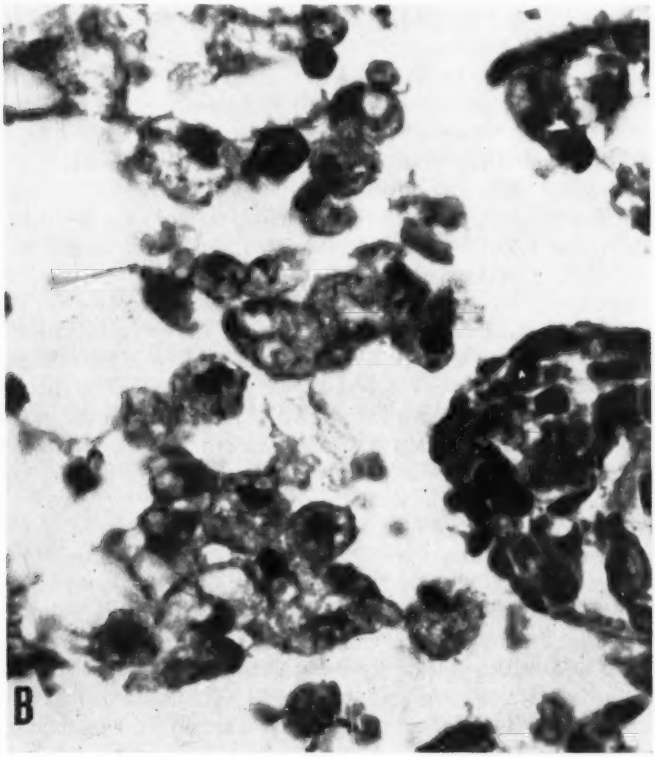

FIG. 4B. Area outlined in Fig. 4A magnified ×800.

was seen about the subarachnoid vessels, but the blood vessels themselves showed little histological change except for mild endothelial hyperplasia.

DISCUSSION

Of the methods that appeared to be available for obliteration of part of the blood supply to the spinal cord, none was capable of producing completely predictable lesions. Unpublished data from this laboratory indicated that occlusion of the aorta for approximately 35 minutes often produced paraplegia which was irreversible. Periods less than this seemed to produce changes which were extremely transient and most frequently could not be detected neurologically in the dog. Injection of particulate matter either produced no lesion whatever or resulted in profound and severe neurological damage which usually brought on the demise of the animal. From injection studies of the vasculature of the spinal canal of the dog, it appeared that the blood supply to the spinal cord was highly variable. If there were a single characteristic feature of the spinal cord in the midthoracic region, it centered about the fact that one or more of the radicular arteries could or did serve as the dominant source of blood. The most obvious course to follow in interruption of this supply was to attack these radicular vessels directly. The mere opening and closing of the dura mater with manipulations that tend to disrupt the arachnoid in itself produces considerable scarring. In the hands of the inexperienced operator, this scarring can be extensive indeed. In one of the preliminary animals when the operation was stopped because it was felt that trauma might have been produced inadvertently, cavitation similar to that previously reported[2] was observed. Since these vessels are isolated with great difficulty, it was concluded that the best approach would be to interrupt them by section of the whole nerve root within the sleeve extradurally. The question then arose as to whether the neural impairment imposed by such generous sectioning of both anterior and posterior nerve roots over an extensive segment of the spinal cord might not in itself result in severe enough neurological impairment that the effect of interruption of the blood supply would be obscured. The variability in neurological deficit found in the various animals would speak rather definitely for differences in vascular supply rather than in the effect of loss of neurons, since the latter would tend to be rather constant. The extension of the effects beyond the limits of the gray matter would also speak strongly for the effects being vascular rather than direct neuronal.

In these experimental ischemic lesions of the spinal cord the most consistent and widespread histological change was loss of neurons in the dorsal horns. This effect usually extended several spinal cord segments above and below the region of experimentation. The neurons of Clark's column were most susceptible to this change. Coexistent with this pattern of neuron loss was a mild gliosis which diffusely involved the gray matter in the ischemic portion of the spinal cord. Usually associated with the neuronal damage was cavity formation in the midline gray matter dorsal to the central canal. The

cavitation was much more restricted to the level of most severe ischemia than were the neuronal changes. The usual cavity appeared to be a passive separation of the tissue reaction. This type of cavity seemed to result from a spatial rearrangement of the tissue subsequent to shrinkage of the dorsal horn gray matter. This passive form of cavitation may be considered the same process as that leading to "hydrocephalus ex vacuo" in the brain. This type of cavitation was usually found in dogs whose functional impairment was mild.

In 3 of the cavitations studied histologically there was actual necrosis of the interstitial as well as the neural elements in the dorsal gray matter and adjacent white matter. These cases presented the usual picture of myelomalacia with active phagocytosis. This seems to represent a more severe ischemia of a sufficient degree to cause necrosis of the traversing long tracts. In conformity to this view, the 3 dogs with this type of lesion had severe and permanent functional impairment of the long tracts as demonstrated by paraplegia. These cases demonstrate that in more severe degrees of spinal cord ischemia myelomalacia as well as shrinkage of the gray matter contribute to the production of central cavitation.

The dominant role of neuron loss in spinal cord ischemia provides experimental support for the emphasis on this type of pathological change in some more generalized diseases of the central nervous system. Scholz[7] reported that loss of "topistic units" of ganglion cells in the brain may be the dominant pathological change and of equal significance to encephalomalacia in some cases of cerebrovascular disease. In the present study the pattern of neuron loss is probably more dependent on circulatory factors than individual differences in susceptibility of neuron types to ischemia.

A consistent feature of the cavitation was its point of origin in the midline gray matter dorsal to the central canal. Whether the vascular interference was directed at the surface vessels, the anterior spinal vessels, or multiple radicular vessels had no effect on the point of origin. In 1 case, a typical small cavity was produced by merely opening and closing the dura mater. Cavitation in this region has also been seen in other experimental spinal cord injuries such as concussion.

While cavitation in the region just dorsal to the central canal is an early histological change in spinal cord ischemia, it does not appear to represent insufficiency of arterial blood supply from any particular vessel. It seems more likely that this lesion results from an impairment in the general circulatory state of the spinal cord. Interference with venous drainage may be the responsible factor. The central portion of the spinal cord is inherently devoid of venous collateral facility. Within the central region, the ventral gray matter is relatively well drained by large veins traversing the anterior median fissure. The posterior gray matter therefore appears most vulnerable to venous insufficiency. Venous blood from this area must drain through the entire substance of the spinal cord to reach the large surface veins. In a state of edema compressing all vessels within the spinal cord substance it

seems reasonable that venous drainage from the posterior central region would be most seriously impeded. Venous insufficiency is also suggested by involvement of the gray matter even in cases in which necrosis of the interstitial elements occurs. In arterial occlusions the interstitial elements of the gray matter are relatively resistant to necrosis.

There is striking similarity between these spinal cord cavitations and those produced by experimental arachnoiditis.[6] The conclusion that vascular factors account for such cavitations is supported by the present investigation. Most of the spinal cord cavitations reported by these investigators, however, suggest a more severe degree of ischemia with necrosis of the long tracts and interstitial neural elements.

SUMMARY

The earliest histological change in mild spinal cord ischemia in dogs is loss of neurons in the dorsal horns. This is usually followed by shrinkage of the posterior gray matter with passive separation of the interstitial neural elements and cavity formation. In more severe degrees of spinal cord ischemia there is necrosis of the traversing long tracts and interstitial elements as well as the neuron loss. This produces a picture of myelomalacia with cavitation. Both types of cavitation involve primarily the central gray matter dorsal to the central canal, but may occasionally extend from this region into the adjacent white matter.

Venous insufficiency secondary to spinal cord edema may account for the pattern of neuron loss and the constant origin of cavitation in the dorsal gray matter. The reasons for this conclusion are discussed.

The advice and assistance in interpretation of Dr. Orville T. Bailey were of inestimable value in the conduct of these experiments.

The photomicrographs were ably prepared by Mr. James F. Glore.

Mrs. Mercedes Guess devotedly assisted in the surgical preparation and care of the animals.

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EXCISION OF OCCLUSIVE LESIONS OF THE MIDDLE CEREBRAL ARTERY*

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(Received for publication September 7, 1955)

When the middle cerebral artery becomes obstructed, the large and important area of cortex to which that vessel is assigned is placed in great peril. Fortunately, there are paths available for readjustment of the circulation so that ischemia is lessened by inflow of blood from branches of the other cerebral arteries. This new circulation is sometimes adequate for recovery, but in other cases is not sufficient and recovery is incomplete or does not occur. Even should function be regained, the danger is not lifted, for the collateral circulation may fail later and the stroke recur. For these reasons, in 2 patients with middle cerebral artery occlusion, an attempt has been made to remove the obstruction and to allow blood flow to be reestablished through its usual path.

CASE REPORTS

Case 1.† The patient was a 47-year-old man who had had a traumatic amputation of the left leg at the midthigh in 1928 and had also been treated in the past for syphilis.

On Sept. 14, 1954, he wakened after 2 hours of sleep feeling lightheaded, and found himself paralyzed on the left side. His tongue felt thick and there was slurring of his speech. Subsequently, there was some recovery in his leg. He was admitted to the hospital 2 days later.

Examination. There was left facial weakness with protrusion of the tongue toward the left. The left arm was flaccid and motionless, with diminished reflexes. There was weakness of flexion of the stump of the left leg. Two-point discrimination and position sense were impaired on the left.

Course. Some improvement occurred, and he gradually regained strength in the stump of his leg and some movement in the arm, but selective weakness of pyramidal distribution with extreme impairment of finger dexterity and blunting of two-point discrimination and position sense persisted.

1st Angiography. On October 8, 1954, arteriograms were made. The films are reproduced elsewhere.[12] There was obstruction of the right middle cerebral artery just beyond the posterior temporal branch and impaired filling in the distribution of the ascending branches. An hour after the arteriogram, his arm became paralyzed again, but some recovery was observed during the next 3 days.

Operation. On Oct. 12, 1954, the right middle cerebral artery was exposed, with

* Read at the meeting of the Harvey Cushing Society, Quebec, Canada, May 18, 1955.

† This case was reported previously in a different connection and the preoperative angiograms were reproduced.[12] The present case is Case 2 of that report.

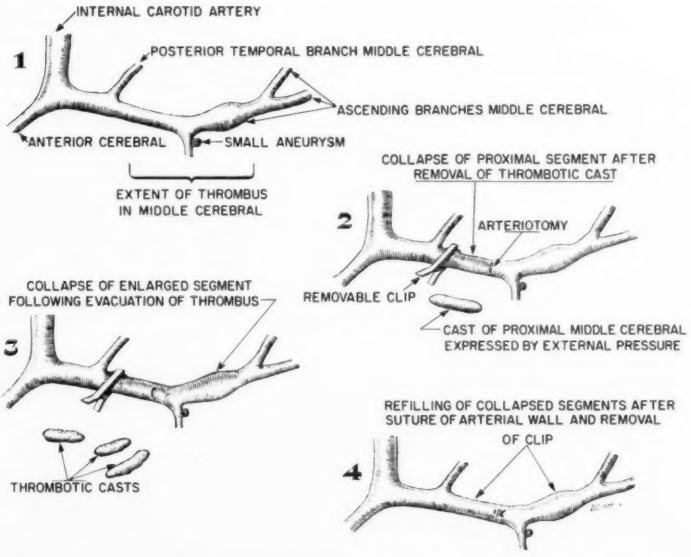
INTERNAL CAROTID ARTERY
1
POSTERIOR TEMPORAL BRANCH MIDDLE CEREBRAL
ASCENDING BRANCHES MIDDLE CEREBRAL
ANTERIOR CEREBRAL
SMALL ANEURYSM
EXTENT OF THROMBUS IN MIDDLE CEREBRAL
COLLAPSE OF PROXIMAL SEGMENT AFTER REMOVAL OF THROMBOTIC CAST
2
ARTERIOTOMY
REMOVABLE CLIP
CAST OF PROXIMAL MIDDLE CEREBRAL EXPRESSED BY EXTERNAL PRESSURE
COLLAPSE OF ENLARGED SEGMENT FOLLOWING EVACUATION OF THROMBUS
3
THROMBOTIC CASTS
REFILLING OF COLLAPSED SEGMENTS AFTER SUTURE OF ARTERIAL WALL AND REMOVAL OF CLIP
4

FIG 1. *Case 1.* Operative findings and steps in operative procedure are outlined.

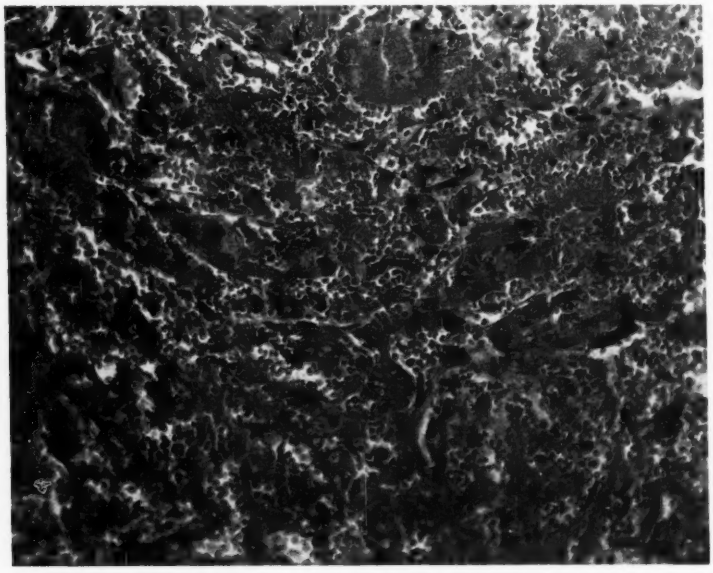

FIG. 2. *Case 1.* Organizing thrombus removed from the middle cerebral artery. Hematoxylin eosin stain, ×225.

hypothermia (28°C.) as an adjunct. There was a segmental thrombus which extended from just beyond the origin of the posterior temporal branch to well beyond the next branching (Fig. 1). The vessel was incised and the thrombus was expressed by external pressure. After the vessel was sewn and the proximal control was released, the occluded segment ballooned up with blood and the wound was closed. The thrombus was an organizing one (Fig. 2), its age by history compatible with the histological appearance.

2nd Angiography. Sixteen days after operation, right carotid angiography was repeated. This time one exposure was made after the injection of only 3 cc. of 35 per cent Diodrast, because of his response after the first arteriogram. The films demonstrated patency of the ascending branches of the middle cerebral artery, though the posterior temporal branch did not fill (Fig. 3).

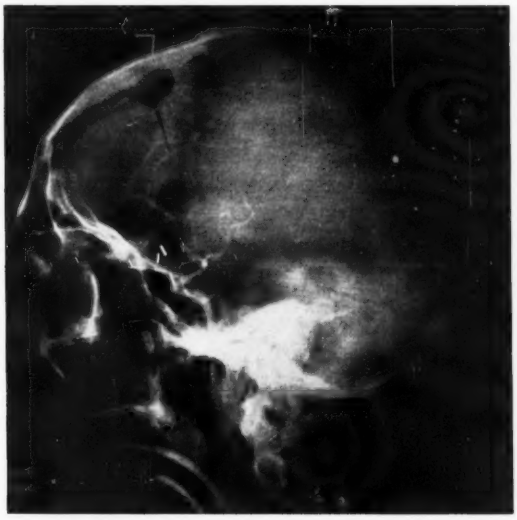

FIG. 3. *Case 1.* Postoperative right carotid angiogram. The ascending branches of the middle cerebral artery are filled but the posterior temporal branch is not.

Course. Clinical recovery has been excellent. He walks as well as ever with his prosthesis. The upper extremity shows very good strength throughout, but there is still slight awkwardness in movements requiring finger dexterity.

Summary. In a 47-year-old man, who had previously had an amputation of the left leg and had also been treated for syphilis, left hemiplegia developed during sleep. Spontaneous improvement began early, but after 3 weeks the neurological deficit was still severe. The lesion was demonstrated by angiography to be an obstruction of the common origin of the ascending branches of the right middle cerebral artery. A thrombus was removed from the vessel, and later arteriography revealed patency of the ascending branches of the right middle cerebral vessel, though the posterior temporal branch, previously open, did not fill. Clinical recovery has been good.

FIG. 4. *Case 2.* Preoperative left carotid angiogram. There is obstruction of the left middle cerebral artery.

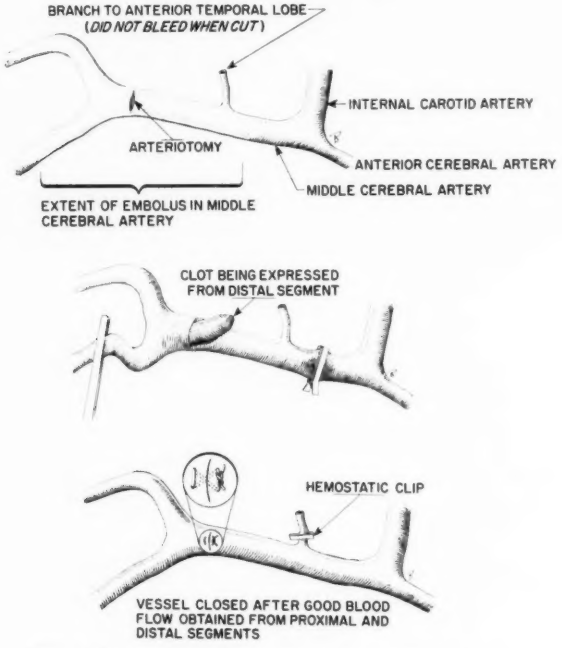
BRANCH TO ANTERIOR TEMPORAL LOBE
(DID NOT BLEED WHEN CUT)
INTERNAL CAROTID ARTERY
ARTERIOTOMY
ANTERIOR CEREBRAL ARTERY
MIDDLE CEREBRAL ARTERY
EXTENT OF EMBOLUS IN MIDDLE CEREBRAL ARTERY
CLOT BEING EXPRESSED FROM DISTAL SEGMENT
HEMOSTATIC CLIP
VESSEL CLOSED AFTER GOOD BLOOD FLOW OBTAINED FROM PROXIMAL AND DISTAL SEGMENTS

FIG. 5. *Case 2.* Operative findings and procedure are shown.

Case 2. The patient was a 51-year-old obese woman who lived alone. On the morning of Nov. 26, 1954, she was discovered in her room with a right-sided paralysis and inability to speak. She was admitted to the hospital at once.

Examination. Her blood pressure was 150/80, her pulse was irregular, and a systolic murmur was heard over the apex of the heart and in the aortic area. There was total paralysis of the right arm and leg, and the right side of the face was weak. She was unable to speak, but was conscious.

Angiography. A left carotid arteriogram was made on the morning of admission. Five cc. of 35 per cent Diodrast were injected, and a single exposure was made. The film revealed good filling of the carotid and anterior cerebral arteries, but the

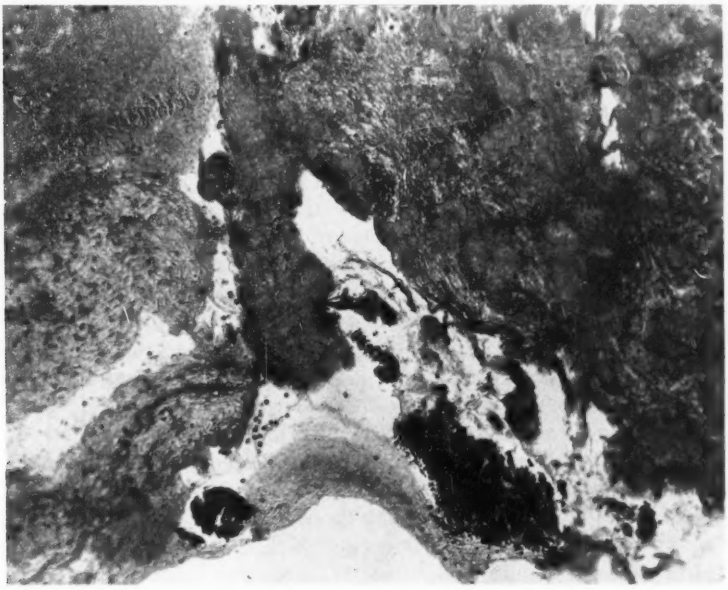

FIG. 6. *Case 2.* Embolus removed from the left middle cerebral artery. A small fleck of calcium is present. Hematoxylin eosin stain, ×100.

middle cerebral artery was filled only in its most proximal part (Fig. 4). The column of dye ended abruptly, and there was no filling in the peripheral territory of the middle cerebral artery.

Operation. That afternoon, operation was carried out, again with hypothermia (29°C.). The internal carotid artery and the most proximal part of the middle cerebral artery had a normal blue-gray color; beyond this, the middle cerebral artery was pale and distended and was firm to palpation. The embolus reached to a distance of approximately 2 cm. beyond the bifurcation of the middle cerebral artery (Fig. 5).

A small opening was made just proximal to the bifurcation of the artery, and it was possible to extract quantities of friable red-yellow material from within the vessel. It was thought that all the embolus distal to the arteriotomy had been removed when good backflow was obtained. An excellent forward flow of blood followed expression of the clot from the proximal part of the artery. The opening in the

vessel was then sewn with a single figure-of-eight stitch, and the wound was closed. The histological appearance of the embolus is illustrated in Fig. 6.

Course. Postoperatively, the patient remained hemiplegic and aphasic. Repeat arteriograms showed the middle cerebral artery obstructed as before.

Summary. A 51-year-old woman had complete right hemiplegia and aphasia caused by an embolus of the left middle cerebral artery from a fibrillating heart. The obstruction was demonstrated by arteriography and removed surgically, but the patient's condition did not improve and repeat angiography showed that the vessel was occluded as before.

DISCUSSION

Following obstruction of the middle cerebral artery or a branch of that vessel there are important and interesting readjustments of the circulation of the brain, and the efficiency with which these are brought about will limit, in a measure, the devastation that results. That extensive softening is not the inevitable result of such an occlusion was recognized many years ago and has been illustrated more recently, both from autopsy data[11] and by the remarkable, even complete, recovery of function achieved by several patients after surgical closure of the middle cerebral artery.[5,7] The question of survival of infarction in the territory assigned that vessel appears not to be absolutely settled from the moment of occlusion, but to depend upon factors that come into play afterward. Among these are propagation of clot proximally into the region of origin of the penetrating branches and the efficiency or inefficiency of the collateral circulation. The surface connections between the terminal branches of the cerebral arteries have been known for three-quarters of a century, but their function as alternative pathways for circulation was subject for debate. More recently, Vander Eecken and Adams[11] have made a precise study of these vessels in normal brains and in brains in which a major vessel was occluded. They have demonstrated that these terminal connections, which they named *meningeal arterial anastomoses*, do indeed serve to protect the cortex from infarction in the territory of distribution of an occluded vessel. They were also able to correlate the protection afforded with the total cross-sectional area of the anastomotic bed, and they suggested that when the meningeal arterial anastomoses are narrowed by disease the protective function might be impaired. The importance of adequately maintained blood pressure in the face of the increased peripheral resistance imposed by the collateral vessels was also discussed by these authors.

The rapidity with which the collateral circulation is brought into play must be very important, but has not been studied. The balance between the state of the general circulation and the efficiency of the surface anastomoses between the cerebral arteries is a precarious one, not only during the period immediately following the stroke but also afterward.[12] It would appear, therefore, that definite advantages might be expected were it possible to

relieve the obstruction and reestablish blood flow through the middle cerebral artery.

Attempts to relieve cerebral ischemia by reopening an occluded vessel have previously been confined to the carotid artery in the neck. The release of deliberate surgical closure made for the treatment of aneurysm has sometimes been followed by great improvement in lost function,[1] so that now it is more or less standard practice to make these occlusions so that they can be released if necessary. In 1951, Spence removed a spontaneously occurring occlusive clot from the internal carotid artery with relief of symptoms, but there was recurrence after 3 years.[9] This approach to carotid thrombosis in the neck was suggested by Fisher in 1952,[4] and several unsuccessful attempts have been recorded since.[8,10] Eastcott *et al.*,[3] have recorded successful reconstruction of the internal carotid artery in a patient with intermittent hemiplegia, with relief of symptoms and arteriographic demonstration of patency.

Meanwhile, interest in the treatment of cerebral aneurysms has resulted in improved methods for exposure and for dealing with cerebral arteries. Particularly pertinent was the demonstration by Campbell and Burklund[2] that the middle cerebral artery can be sewn. Also of great importance was the development of hypothermia as an adjunct to surgery and its application to surgery of the blood vessels of the brain.[6]

Perhaps because recognition of cerebral softenings antedated recognition of arterial obstruction as their cause, emphasis and interest have centered much more upon cerebral infarction than upon arterial obstruction. As a result, precise information regarding sites of election of occlusive lesions and the natural history of the various occlusions is badly needed. Until the natural history of middle cerebral closure is better known, it will not be possible to be precise in the evaluation of treatment, and certainly before direct surgical approach to intracranial arterial obstruction can be appraised, a considerable experience with this method must be available for analysis.

SUMMARY

Occlusive lesions have been removed from the middle cerebral artery in 2 patients. The first was a 47-year-old man who had shown some recovery from hemiplegia after a right middle cerebral artery thrombosis. Four weeks after onset, the right middle cerebral artery was incised, the thrombus was removed, and the vessel was sewn. Subsequently, the ascending branches of the middle cerebral artery were demonstrated by arteriography to be open. There has been recovery of function except for finger dexterity. The second patient was a 51-year-old woman who had auricular fibrillation. She was studied and operated upon 12 hours after being found with right hemiplegia and aphasia. An embolus was excised from the left middle cerebral artery. Her neurological status has not improved, and subsequent arteriography revealed, as before, obstruction of the middle cerebral vessel.

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EXPERIENCES WITH HIGH CERVICAL CORDOTOMY

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(Received for publication September 16, 1955)

THE development of anterior-lateral cordotomy has been covered by many excellent reviews.[1,2,5,8] The results of cordotomies performed in the high thoracic region have given relatively satisfactory results for the relief of intractable pain in the trunk, pelvis, and lower extremities. However, two disadvantages of cordotomy at this level have existed: the level of analgesia obtained by this method has not always remained sufficiently high to provide continued relief of pain, and the retention of "islands" of intact sensation distal to the cordotomy have in many instances negated an otherwise satisfactory result. In order to overcome these disadvantages, other surgical procedures have been employed. Among these are prefrontal lobotomy, mesencephalic tractotomy and medullary spinothalamic tractotomy. These procedures have not gained widespread utilization because of technical difficulties inherent with the operation, relatively high mortality rates and inconstancy of results. At the present time, the indications for these operations are considered to be rather sharply limited, and in general they are not considered to be the operation of choice for the relief of intractable pain below the face. The most notable exception to this is in the instances of intractable pain associated with severe narcotic addiction and preexisting mental changes. For these patients prefrontal lobotomy has been of considerable value.[3]

As a result of the frequent unsatisfactory results obtained with high thoracic cordotomies and the disadvantages encountered with other neurosurgical procedures for relief of pain, cordotomy done in the high cervical region has enjoyed increasing popularity.[4,6,7] High cervical cordotomy offers the advantages of relatively few technical difficulties, sustained high levels of analgesia without "islands" of hypalgesia or sparing distal to the site of the cord incision and a relatively low operative mortality rate. The purpose of this paper is to present and discuss our experience with 81 consecutive cases of high cervical cordotomy performed by the members of the neurosurgical service of the University of Minnesota Hospitals during the years 1948 through 1954.

When the program of performing high cervical cordotomies was first

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instituted in this clinic, only those patients with intractable pain in the upper thorax, brachial plexus, and upper extremity were subjected to this procedure. However, over a period of years it has been felt more and more strongly that high cervical cordotomy was the operation of choice for patients with intractable pain in the trunk, pelvis, and lower extremities and because of this, many patients with pain in these locations have been subjected to high cervical cordotomy and are included in this study.

Initially, the selection of these cases for surgery was not based on a rigid set of criteria and some of the patients were desperately ill and debilitated when first seen as possible candidates for surgical relief of pain. Experiences with patients in this category have been most disappointing and it must be stressed that the optimum time for cordotomy is prior to the extreme general physical decline and severe narcotic addiction, for these attend a much

TABLE 1

Level of incision	C1	C2	C3	C4	C5	C6	C7	C8	T1	T2	
Number of cordotomies	6	71	5	4	1	2	3	1	9	10	Total 112

slower postoperative convalescence and a higher operative mortality rate.

In this series of 81 cases there were 11 operative deaths, an operative mortality rate of 13.5 per cent. Two of these patients, obviously extremely poor surgical candidates, suffered cardiac arrest prior to making the incision in the cord. They are included in the mortality statistics but for obvious reasons are not included in the other statistics presented here. The remaining 9 patients were terminally ill and debilitated and were considered poor surgical risks.

Included in this series of 81 patients with high cervical cordotomy are 33 patients upon whom bilateral incisions were made to relieve either midline or bilateral pain. In 14 patients the bilateral incisions were done in the higher cervical levels and in 19 the high cervical incision was combined with a high thoracic incision. Thus, a total of 112 cordotomies were performed in this series of 81 cases (Table 1).

The etiology of the pain varied but predominantly it was caused by neuritis or radiculitis secondary to local or regional extension of malignancy. Among the other diseases encountered in this study were tabes dorsalis, phantom limb pain, leukemia and osteoarthritis of the spine (Table 2).

SURGICAL TECHNIQUE

The upright or sitting position was used for all patients in reasonably good physical condition upon whom unilateral high cervical cordotomies were done. At first, this position was used for bilateral procedures but because of the associated drop in blood pressure and ensuing complications it became obvious that a horizontal or a prone position must be used whenever doing bilateral cordotomies. Three of the deaths in this series perhaps can

be related to doing bilateral procedures with the patient in the upright position.

Of the 81 cases, in 27 local anesthesia was utilized and in 54 general anesthesia was used. Many of the earlier operations were performed under local anesthesia with testing for the level of analgesia during section of the spinothalamic tract within the cord. However, it is our opinion that the use of local anesthesia with immediate testing of the patient is not necessary to achieve the desired level of analgesia. In fact, it is considered to be very unreliable and at times misleading. Better results are obtainable using general anesthesia.

A wide hemilaminectomy of the 1st and 2nd cervical vertebrae is done. Beginning in the midline the dura mater is opened in a horseshoe fashion with the limbs of the

TABLE 2

Classification of diseases

Disease	Number of Cases
Carcinoma, female genitourinary tract	16
Carcinoma, male genitourinary tract	12
Carcinoma, breast	7
Carcinoma, lung	8
Carcinoma, pancreas	7
Carcinoma, gastrointestinal tract	11
Amputation stump pain	6
Tabes dorsalis	5
Sarcoma, hip joint	2
Malignant melanoma	2
Neurofibroma, sacral plexus	1
Osteoarthritis, spine	1
Chondrosarcoma, pelvis	1
Leukemia	1
Carcinoma, primary unknown	1
Total	81

incision carried far laterally, thus forming a dural flap. This is retracted and secured to the adjacent muscles with silk sutures. This is an important step for it permits good visualization of the anterolateral section of the cord with a minimum of rotation. The arachnoid is open along its lateral margin at the site of the cordotomy. The posterior rootlets of the 2nd cervical nerve on the side of the cordotomy are sectioned. The attachment of the 2nd dentate ligament is grasped with fine hemostatic forceps and severed lateral to the forceps. The 1st dentate ligament is occasionally cut at its attachment, but more commonly it is cut across transversely. This permits adequate rotation of the cord without alteration of the structures above the 1st cervical level. Occasionally the 3rd dentate ligament is detached. The cord is gently rotated by traction on the 2nd dentate ligament and a cataract knife is inserted for a depth of 4 to 5 mm. in the cord beginning at the dentate ligament and swept forward to beyond the emergence of the anterior rootlets. Instead of making the entire incision with the knife, a forceps is used to crush the most medial and

anterior areas, thereby obviating possible section of the anterior spinal artery or its major branches. The section is usually made opposite the rootlets of the 2nd cervical nerve but may be made as high as the 1st cervical nerve when the operation is done with the patient in a sitting position.

RESULTS

Eighty-six cord sections were performed between the levels of C1 and C4, 71 being performed at C2. Immediately after the operation the level of analgesia was as high or higher than the 6th cervical segment in 73 of the 86 cases. Of these 73 cases, in which the postoperative follow-up varied from 1 month to 2 years, in only 1 was there a drop in analgesia of more than two segmental levels. This patient with an original level at C4 was down to T2 10 months following cordotomy. Four patients with an initial level of analgesia at C2 subsequently dropped to C4. Sparing or "islands" of hypalgesia was observed in 5 cases. Despite this the patients obtained complete relief of pain.

For bilateral cordotomies the second incision was made at T1-T2 in 19 cases. All of these patients exhibited an immediate postoperative level of analgesia at T10 or higher. Eighteen of these patients have been followed from 1 month to 2 years. In this group, 5 patients had a drop of at least four segmental levels of analgesia and after 2 months, 3 others had no demonstrable level of analgesia. Thus it is apparent that the level of analgesia obtained with high cervical cordotomy unquestionably tends to fall less than after high thoracic cordotomy.

It is easier to establish a level of analgesia nearer to the site of the cord section in high cervical cordotomies than in high thoracic cordotomies. This can be explained in part on a neuro-anatomical basis. The fibers comprising the spinothalamic tract in the high thoracic region are not grouped together in as compact a bundle as in the high cervical cord thereby making it more likely for some of the tract to be spared by the incision. This may account in part for the differences in the immediate and subsequent drops in levels of analgesia. In this series 73 of the 86 cordotomies done between C1 and C4 resulted in an initial level of analgesia within two segments of the site of the cordotomy. Comparing this with the 19 thoracic cordotomies in this series, there were 2 cases in which there was an immediate level of analgesia within two segments of the site of the section, 5 within four segments of the section, and 12 that were six or more segments below the site of the cordotomy.

Bilateral high cervical cordotomy was done in 14 cases (Table 3). In 12 it was performed in one stage. There were 5 operative deaths in these 12 cases. In 4 of the fatalities there was a bilateral level of analgesia established up to the 4th cervical segment and in the fifth case, up to the 4th cervical segment on one side and to the 5th thoracic segment on the other side. Nine patients survived bilateral high cervical cordotomy; 7 of the operations were one-stage, and 2 were two-stage procedures. The highest level of analgesia obtained in these surviving patients was C4, T4. The obvious con-

clusion is that bilateral high cervical cordotomy is fatal if the level of analgesia obtained is high enough to relieve bilateral upper thoracic and lower cervical pain. It must be admitted that these patients were the most debilitated and cancer-riddled of the series. They were the ones who had extensive bilateral pain necessitating high levels of analgesia bilaterally. There is, of course, the theoretical possibility that patients in better general condition might tolerate such an operation. Such patients with intractable pain were not seen in this series.

Postoperative paresis of the ipsilateral extremities was observed in 13 cases. This disappeared within a matter of a few days in all but one case. In

TABLE 3

Bilateral cervical cordotomies

Case Number	Sites of Cordotomy	Levels of Analgesia		Results of Operation
		Immediate	Eventual	
10	C2—C2	C4—C4	C4—C4	Two-stage operation; 2-week interval. Expired 2nd day after 2nd stage
11	C2—C7	C4—T5	C4—T5	One-stage operation. Expired 2 days after surgery in hepatic coma
14	C2—C6	C4—T4	C4—Unknown	One-stage operation. Complete relief of pain
16	C2—C7	C4—T4	C4—T4	One-stage operation. Complete relief of pain
17	C2—C4	C4—C7	C4—C7	One-stage operation. Expired 3 days after operation
19	C2—C4	C5—C6	C5—T1	Two-stage operation; 3 week interval. Complete relief of pain
20	C2—C6	C4—T4	C4—T4	One-stage operation. Complete relief of pain. Permanent urinary retention
23	C1—C7	C4—T2	C4—Unknown	One-stage operation. Complete relief of pain
24	C1—C4	C4—C6	C4—C6	One-stage operation. Complete relief of pain
37	C2—C5	C5—T2	Unknown	One-stage operation. Complete relief of pain
40	C2—C4	C4—C4	C4—C4	One-stage operation. Complete relief. Expired 24 hrs. after operation
41	C2—C3	T2—no level	T2—T8	One-stage operation. Cordotomy repeated. Complete relief 6 weeks. Then recurrence of pain
55	C1—C3	T3—T8	T3—T8	One-stage operation. Complete relief of pain
79	C2—C8	C3—T7	C3—T7	One-stage operation. Complete relief of pain

this instance while the weakness persisted, it was not severe and it was not disabling to the patient. Transitory paralysis of the ipsilateral intercostal muscles and diaphragm occurred in almost all of our cases. This usually disappeared within 24 hours and has not caused pulmonary complications in any instance. It has been observed that patients who exhibited this transitory paralysis of the respiratory muscles are more likely to obtain a high permanent level of analgesia without subsequent drop or "islands" of hypalgesia.

Urinary retention was observed in 11 patients, all of whom had bilateral cordotomies. In 2 cases it was permanent, requiring continuous catheter drainage of the bladder. In the other 9 cases, however, it disappeared en-

tirely in a matter of a few days to 3 weeks. Both patients with permanent urinary retention had bilateral cordotomies with levels of analgesia established at C5-T6 and C4-T4 respectively. Perhaps this frequency of urinary retention would have been higher had not many of the patients already had urinary shunting procedures because of the nature and extent of the cancer.

Seven patients with amputation stump pain and phantom limb pain had high cervical cordotomies. Five of these patients had amputations of the lower extremity and 2 had amputations of the upper extremities. Following high cervical cordotomy 4 of these patients obtained complete relief of pain. Three patients were not completely relieved of their pain but in every case its severity was lessened. It is of interest to note in all cases in which the relief of pain was complete the patient had a permanent C4 level of analgesia without "islands" of hypalgesia. Whereas, in the 3 cases considered as failures, one patient had an island of sparing over the amputation stump; one had a drop in level of analgesia to T9; and the third patient had been subjected to previous rhizotomy from C4 through T4, sympathectomy and prefrontal lobotomy without change in the pain. As a result of these previous procedures, it was almost impossible to evaluate the postoperative relief. However, this patient is considered not relieved. On the basis of these results it is felt that amputation stump pain and phantom limb pain can be relieved by high cervical cordotomy provided the level of analgesia is high and no "islands" of sensation remain.

SUMMARY

1. Eighty-one consecutive cases of cordotomy in the high cervical region, performed at the University of Minnesota Hospitals, have been reviewed.

2. Patients with intractable pain should be considered as candidates for the surgical relief of pain prior to physical debility and narcotic addiction.

3. Cordotomy in the high cervical region is considered to be the treatment of choice for the relief of pain below the face because of the infrequency of technical difficulties with the procedure, relatively low mortality rate, and sustained high levels of analgesia without "sparing" distal to the site of the cord incision.

4. Paresis of the ipsilateral extremities occurred in 13 cases. In 12 it was transient and in the remaining instance it was not disabling.

5. Urinary retention was observed in 11 cases. In only 2 was it permanent. In all 11 cases it occurred following a bilateral cordotomy.

6. Amputation stump pain was relieved if the level of analgesia was sufficiently high and if there were no "islands" of sparing distal to the cord section.

7. There were 11 operative deaths in this series of 81 cases, a mortality rate of 13.5 per cent. With more careful selection of cases and the omission of the "sitting up" position for bilateral cordotomies, it is felt that this operative mortality rate can be very significantly lessened.

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THE MYELOGRAPHIC APPEARANCE OF SACRAL CYSTS

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(Received for publication September 16, 1955)

THE presence of cysts within the sacral spinal canal was first described as an incidental autopsy finding by Tarlov[9] in 1938. More recent reports indicate that these structures may be the cause of pain and neurological disturbances.[4,6–8,10–12] Since only 16 cases were found in the medical literature, we would like to report our clinical and roentgenographic experience with 4 patients.

During a study of the filum terminale in 30 routine autopsies, Tarlov[9] found 5 cysts attached to the posterior sacral and coccygeal nerves. During life one of these patients had exhibited signs and symptoms referable to the cyst. In 1948[10] he described a patient with sciatic pain who was cured by removal of a sacral cyst. Although a myelogram had not been done, he felt that it probably would have been negative since the cyst was 1 cm. distal to the origin of the nerve root from the sacral dural sheath and was not in continuity with the subarachnoid space. In a second series of 60 cadavers he found cysts in 8 cases.[11]

In 1950 Weiford[12] described a patient with pain in the sacral area associated with bladder incontinence. At operation, a midline cyst was disclosed arising at the tip of the dural sac in the region of the filum terminale. No communications with the subarachnoid space could be demonstrated although a myelographic examination was not performed.

The first myelographic visualization of a sacral cyst was reported by Schreiber and Haddad in 1951.[6] In their patient a collection of Pantopaque beneath the right upper sacrum and distal to the usual termination of the dural sheath was noted at the time of the myelographic examination. There was also thinning of the bone overlying the cyst. Since then, single case reports of successful myelographic demonstrations of sacral cysts have been described by Taheri *et al.*[8] and Jacobs *et al.*[4] Strully and Heiser[7] reported 5 cases including one in which the cyst was not visualized, but produced an extrinsic pressure defect on the column of contrast material.

CASE REPORTS

Case 1. W.V.T., a 38-year-old white female, had complained of low back pain at various intervals for the past 12 years. For several months, she had had severe pain in her right hip radiating down the posterior aspect of the right thigh, and calf to the lateral aspect of the foot.

Examination. Knee jerks and ankle jerks were active and equal. There were no sensory changes. Minimal to moderate pain was produced on the right side by the straight leg raising test. The pain was increased by coughing, sneezing, and straining, and was relieved by lying down. Roentgenograms of the lumbar spine revealed a spondylolisthesis of the 5th lumbar vertebra with minimal forward slipping, and enlargement of the sacral canal in the region of the 2nd sacral segment.

Lumbar myelography was performed and contrast material was seen to fill a small pocket just to the right of the midline at the level of the 2nd sacral segment which was distal to the normal termination of the dural sheath (Fig. 1). This was felt to represent an arachnoid cyst.

Operation. A laminectomy was done. On removing the bone over the right side of the sacrum a bulging cyst, about 4 cm. in diameter, was disclosed which extended

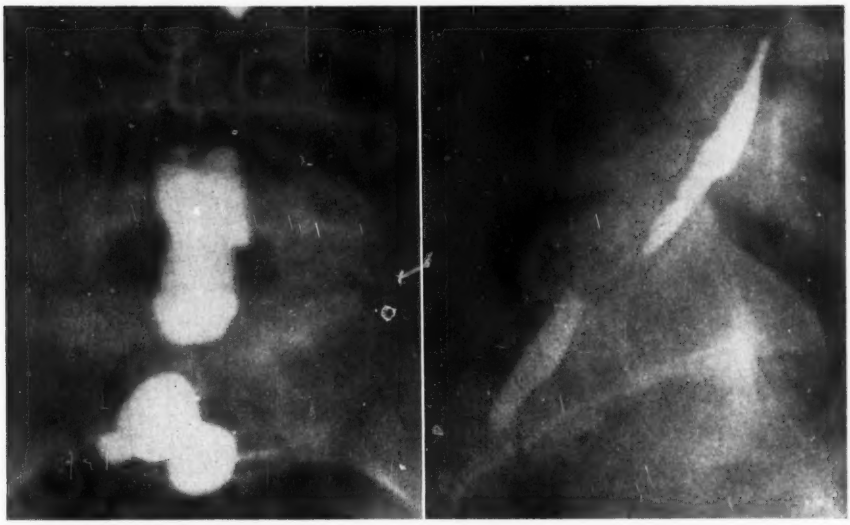

FIG. 1. *Case 1.* AP and lateral views of a sacral cyst lying distal to the usual termination of the dura mater. Since it fills with opaque material there is communication with the subarachnoid space.

below the 2nd sacral segment. It had hollowed out a cavity in the sacrum and was compressing the sacral nerve roots anteriorly. The cyst contained clear fluid plus some residual Pantopaque. It was necessary to take off most of the right half of the upper two sacral segments in order to completely uncover this cyst, which was closely adherent to some of the nerve roots. It was dissected out and traced down to a narrow neck attached to the dural sac at the emergence of the 2nd sacral root. The neck was ligated and the sac was removed. The cyst was considered to be of arachnoidal origin arising near the point of emergence of the 2nd sacral root.

Course. The patient did well postoperatively with complete relief of pain.

Case 2. K.M., a 44-year-old white male, entered the hospital with the chief complaint of back pain of 17 years' duration. Seventeen years before he had fallen from a cliff and had sustained a back injury that had necessitated confinement to bed for

1 month. Since then he had had intermittent episodes of low back pain. The pain was usually brought on by "straining," aggravated by coughing and sneezing and frequently was of such severity that he was forced to go to bed. He had been incapacitated for the past 9 months. The pain did not radiate. A myelogram done 3 months previously was said to have demonstrated a disc at the L5-S1 interspace and a second one at the L4-L5 interspace.

Examination. There was no evidence of atrophy, motor weakness or sensory changes. Straight leg raising tests produced pain only when the lower extremity was brought to a 75°–90° angle. There was some limitation of forward bending and evidence of muscle spasm. Tendon reflexes were equal and active. It was the im-

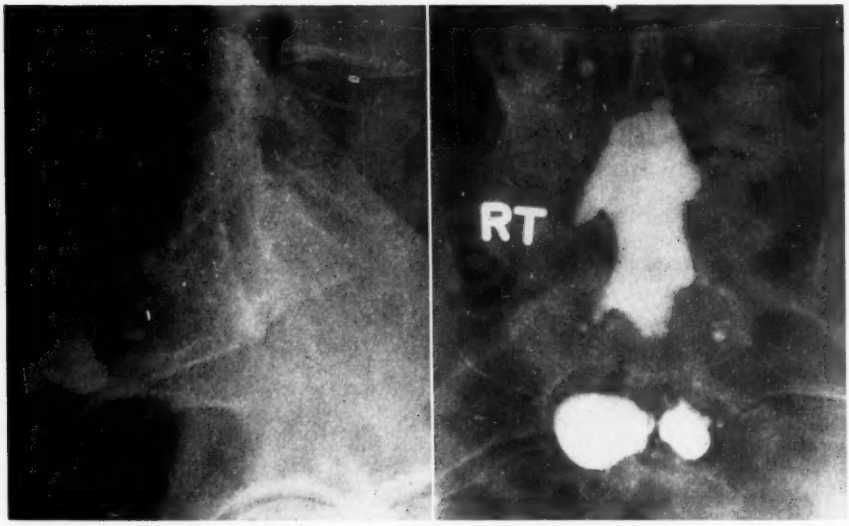

FIG. 2. *Case 2.* AP and lateral views of the sacrum showing the oval area of radiolucency in the midportion of the sacral body and two encysted collections of radio-opaque material. The myelogram demonstrates lack of communication of subarachnoid space with the sacral cysts. There is slight flattening of the opaque column at the level of S1 with incomplete filling and elevation of the axillary sheath.

pression of the examiner that the history suggested a ruptured intervertebral disc, but was not confirmed by the physical findings.

A lateral roentgenogram of the sacrum revealed minimal narrowing of the L5-S1 interspace. An ill-defined oval area of radiolucency, measuring 3×4 cm., was present in the body of the sacrum in the area of the 2nd and 3rd sacral segments. There were two separate collections of residual opaque material in the lower portion of the sacral canal about 2 cm. below the termination of the dural sheath (Fig. 2). The radiolucent area seemed to be divided into two sections with the radio-opaque material lying in the more distal compartment. This material seemed to be encysted since it did not move on tilting the patient in either direction. Although a communication between the cyst and the subarachnoid space must have been present at the time of the original myelogram 3 months before, we were not able to demonstrate a communication. There was slight flattening of the opaque column at the level of

S1 on the left with incomplete filling and elevation of the axillary sheath (Fig. 2). These findings were thought to be consistent with the presence of two arachnoidal cysts in the lower portion of the sacrum without communication with the subarachnoid space.

Operation. It was apparent that the bone overlying the 3rd sacral segment was thinned. Removal of this bone exposed a cystic mass that extended inferiorly to the 4th sacral segment and slightly across the midline. There were many extradural veins that made dissection difficult. A second cystic mass was found which extended anteriorly beneath the dural sac up to the 1st sacral segment. Both of these cysts were attached to the 3rd sacral nerve root. The wall of the cyst seemed to be composed of arachnoid bulging out through the dural sleeve of the nerve root. The dural sleeve was so completely surrounded by this arachnoid that it was impossible to find the neck of the sac. In order to completely remove the cyst, the right 3rd sacral root had to be sacrificed. A 4×5 cm. cavity remained in the anterior part of the sacrum. At no point was the bone eroded anteriorly.

Course. The postoperative convalescence was uneventful.

Pathological Report. The gross specimen consisted of a small fragment of tissue about 1 cm. in its greatest diameter and 4 mm. in thickness. One surface was covered by relatively smooth membrane, the other was dark and hemorrhagic. Microscopic examination by Dr. David E. Smith revealed that the tissue was composed of a regular mass of fine fibrous membranes, blood, and a little adipose tissue that had no distinguishing histologic characteristics.

FIG. 3. *Case 3.* Lateral view of the sacrum demonstrating bony expansion and an oval area of radiolucency. Injection of Pantopaque demonstrates the latter to be the site of a large sacral cyst.

*Case 3.** R.E.F., a 29-year-old white male, was admitted to the hospital for a hemorrhoidectomy. During the process of performing a caudal anesthesia, a cyst containing 15 cc. of fluid was discovered in the lower sacrum. Roentgenograms were taken and revealed an oval area of radiolucency in the sacrum. Injection of Pantopaque demonstrated this to be the site of a cyst (Fig. 3). No communication between the cyst cavity and the subarachnoid space could be demonstrated although the lower portion of the dural sac was displaced laterally by the cyst (Fig. 4).

Examination. There were no abnormal neurological findings and the patient was completely asymptomatic.

Operation. The sacral canal was unroofed, revealing a large cyst containing yellow fluid. A second smaller cyst containing clear colorless fluid was attached to the wall of the larger cyst but no communication was demonstrated between them. The sacral nerve roots coursed anterior and lateral to the cyst. Both cysts were completely excised.

* Courtesy of Dr. Henry G. Schwartz.

Case 4. F. B., a 65-year-old white female, entered the hospital because of difficulty in walking of 5 months' duration. The patient, who was a known hypertensive of 5 years' duration, had experienced a sudden onset of heaviness of the left leg and partial paralysis, associated with episodes of ataxia.

Examination. Blood pressure was 200/100. She walked slowly with a wide-based gait. Neurological findings were normal except for an absent left ankle jerk.

Because of a heavy sensation in the arms a cervical myelogram was performed and found to be negative. At the time of the myelography, the lumbar and sacral areas were examined, and a sacral cyst communicating with the tip of the dural

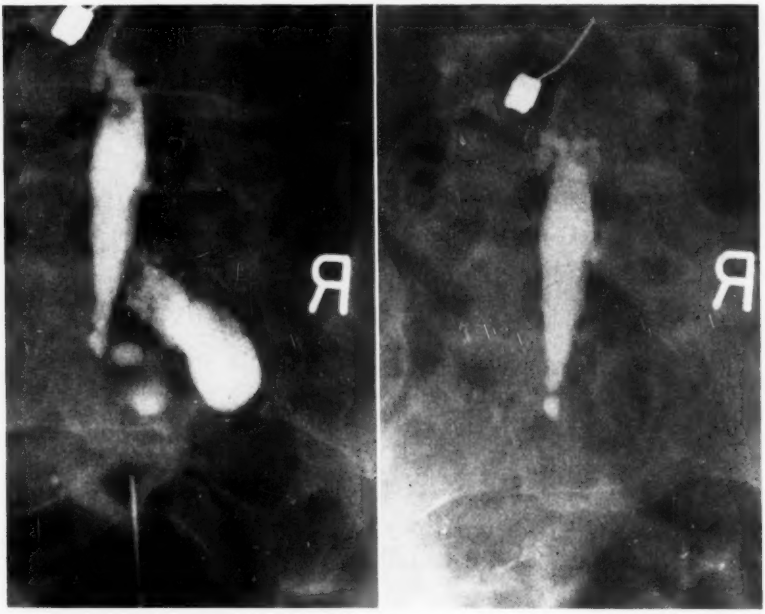

FIG 4. *Case 3.* Myelograms showing lack of communication between the cyst and the subarachnoid space although there is lateral displacement of the cul de sac.

sheath was found (Fig. 5). Since this lesion did not adequately account for the patient's difficulties, it was felt to represent an incidental finding of no clinical significance and exploration was not advised.

DISCUSSION

The origin and cause of sacral cysts are obscure. Tarlov[9] called them perineurial cysts because he believed they arose from a splitting of the nerve root sheath with distention of the potential space between endoneurium and perineurium to form a cyst. They usually occur at the junction of the posterior root and the dorsal ganglion. They almost always occur on the sacral or coccygeal roots, but most commonly on the 2nd and 3rd sacral roots. Tarlov[11] mentioned 2 cases in which cysts were encountered at autopsy

on the thoracic dorsal ganglia. Schreiber and Haddad[6] felt that local trauma was an etiological factor and postulated a progressive sequence of nerve trauma, intraneural hemorrhage and eventual cystic degeneration. Rexed,[5] in an autopsy study, noted proliferation of arachnoid within the tubular dural sheaths surrounding the spinal nerves as they enter the intervertebral foramina. In certain instances, the arachnoidal proliferations invaded the interior of the root fascicles and formed tiny cyst-like cavities. He noted all gradations from a just perceptible increase of arachnoid in the dura mater surrounding the root to an intense proliferation and cyst formation. He could find no evidence of inflammation, neoplasm or degenerating nerve fibers. Most authors feel that these cysts probably should be distinguished from the extradural cysts, originally described by Elsberg, Dyke and Brewer.[3] Similar cysts have been described by both Baker and Webb[2] and Archer *et al.*[1] except that these authors called them intrasacral meningoceles. Regardless of the exact etiology, the gross and microscopic pathology of their cases was similar to ours.

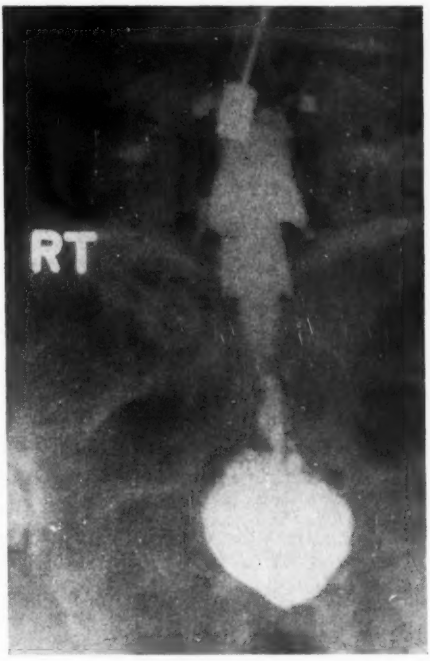

FIG. 5. *Case 4.* Sacral cyst communicating with the caudal extremity of the dural sac. This was an incidental finding in a patient with no symptoms referable to this area.

The chief roentgenographic abnormality associated with this condition consists of thinning of the bone surrounding the sacral canal, usually at the 2nd, 3rd and 4th sacral segments. This can usually be demonstrated only by a lateral view of the sacrum (Figs. 2 and 3). Evidence of sacral bone erosion was present in 3 of our 4 cases. Myelography may only reveal a deformity of the lower end of the thecal sac (Fig. 4). If the cyst communicates with the subarachnoid space, it may be filled with the contrast material. This occurred in 3 of our cases and in 5 cases described in the literature.[6–8]

The clinical significance of these cysts is difficult to evaluate. In most of the cases reviewed by Tarlov[11] the cysts were associated with sciatic pain. However, 2 of our patients were asymptomatic and the cyst was an incidental finding. It may be that cysts that do not communicate with the subarachnoid space are more likely to cause symptoms and as the fluid accumulates they enlarge, become tense, and press on adjacent nerves. On the

other hand, one of the largest cysts in our series which did not communicate with the subarachnoid space occurred in an asymptomatic patient.

SUMMARY

1. The myelographic appearance of 4 patients with sacral cysts has been described.

2. Communication with the subarachnoid space permitting the cyst to be filled with contrast material was present in 3 of the 4 cases.

3. Sacral cysts may be present in asymptomatic patients or may be associated with low back pain.

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A SURVEY OF THE NEUROLOGICAL RESULTS OF 858 SPINAL CORD INJURIES

A COMPARISON OF PATIENTS TREATED WITH AND WITHOUT LAMINECTOMY

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(Received for publication September 26, 1955)

THE literature since World War II contains many articles on the treatment of spinal cord injuries. While most authors agree in general upon proper methods of treating the complications of paraplegia, one apparently unsolved question still remains: "When is laminectomy indicated?"

There seems to be uniform agreement that an exploratory laminectomy is required in patients with penetrating wounds of the spine. A wide difference of opinion exists, however, concerning the indications for laminectomy in cases of recent closed trauma. Most authors consider progressive neurological decline as an absolute indication for intervention,[11] and some feel that most acute spinal cord injuries require laminectomy.[2,3,6,18] In this regard, Tarlov[15,16,17] held it reasonable to assume that cord function may recover provided decompressive measures be instituted before irreversible changes occur. Contrastingly, many observers felt that indications for laminectomy are uncommon in the early post-injury period.[4,5,8,9] Characterizing this opinion is a statement of Mayfield who "rarely finds it necessary to perform a laminectomy," feeling the procedure to be indicated only "(1) if it can be shown that bony compression is retarding the recovery of patients with partial cord lesions and (2) if there is demonstrable obstruction of the spinal canal, if x-ray evidence shows sufficient deformity to cause cord compression and if the clinical findings harbor doubt as to whether the lesion is incomplete." Additionally, Munro suggested that "it is outrageously dangerous" to make exploration in recently injured patients with cervical lesions. Yet, Wannamaker reported no fatalities among 42 patients operated upon under such circumstances.[18]

The presence or absence of subarachnoid block, treated lightly in most reports concerning open injury, is considered by many observers to be a critical factor in assessing the need for operative intervention in patients rendered paraplegic by closed trauma. Davis stressed[4,5] the importance of

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edema of the cord as a contributing factor to paraplegia in the early days following injury and is strongly influenced to operate when spinal subarachnoid block exists. Munro[8,9] supported Davis' views on edema, but appears to delay surgical exploration until it becomes evident that proper conservative management will be ineffective.

Schneider,[13,14] in a recent series of articles, has described two distinct traumatic cervical cord syndromes and carefully outlined indications for intervention. The first, occurring in hyperextension injuries of the cervical spine, and in the absence of subarachnoid block, is a central cord syndrome, which Schneider stated contraindicates surgery. The second, occurring in flexion injuries, also in the absence of subarachnoid block, is an anterior and lateral cord syndrome, which he feels is remediable in the acute phase by section of the denticulate ligaments in accordance with the theory of Kahn[7] on the role of these ligaments in anterior cord compression.

The reported indications for late laminectomy are equally controversial. One opinion, characterized in the publications of Rand,[12] suggests that delayed intervention, particularly after more than 1 year, is seldom followed by significant recovery of function, although the morale of the patient may be improved after exploration. Opposingly Campbell and Meirowsky[2] have noted satisfactory return of function, particularly that mediated by nerve roots, when operation was performed as long as 5 years after injury, and are prone to consider late intervention in the hope of achieving some such benefit for the patient.

The large number of patients studied in the paraplegia service at this hospital afforded an unusual opportunity to assess these problems in the management of spinal cord injuries. That these people were injured in widely separated areas and in many different ways and treated by many physicians by various methods added to, rather than detracted from, the desirability of the material so supplied for study. Records were available concerning the nature and extent of the traumatic process as well as the treatment applied in 858 such cases. This information, supplemented by neurological evaluation of progress since injuries, permitted comparative analysis of the several therapies employed. This analysis forms the subject matter of this report.

MATERIALS AND METHODS

All 858 patients included in this study were regularly admitted to the paraplegia service of this hospital during the past 9 years. In every instance spinal cord injury, either open or closed, had resulted from acute trauma usually rendering the individual essentially completely paraplegic. The records on all patients so admitted (1020) were reviewed, no case being excluded from the series for reasons other than those described below. The primary requirements for inclusion in the group were as follows:

(1) Of critical importance was the availability of a satisfactory description of the original injury, the condition of the patient during the acute and

convalescent stages of his illness, and the type of treatment instituted. Such documentation was available either from the transfer-records of individuals injured while in active service in the armed forces (or treated initially in a service institution) or from communication from the attending physician.

(2) Each patient had a complete record of an adequate evaluation by the Chief or Assistant Chief of Paraplegia at this hospital, regardless of the length of time that had elapsed after injury. No patient was included in the study, however, whose traumatic episode was less than 9 months old.

As the review was begun it was hoped that sufficient information would be available from the records to determine the relative severity of cord damage, and that this could be correlated with recovery or prognosis. It became apparent, however, that radiographic data were not well documented, especially on combat injuries. Operative findings were frequently well described, but on many occasions even gross observation of the cord did not afford a good idea of the prognosis. Thus, many patients operated upon early with no grossly apparent cord damage, failed to recover neurologic function even several years after operation, and others with cords obviously contused or compressed had significant return of cord function. Reports of subarachnoid studies were commonly deficient, but when documented, such information was evaluated.

As the essential factor for evaluation was the degree of recovery exhibited by each subject it was necessary to define arbitrarily categories in which to consider improvement. In the interests of objectivity it was felt that this assessment could best be made primarily on an anatomical basis applying where possible such evidence of neural recovery to clinical improvement. Recovery in neural systems mediating somatic sensory and motor transport, as well as autonomic (bladder) function, was evaluated, therefore, according to the following arbitrary scale:

Grade 0 Recovery.—The modality examined in follow-up review showed less than 2 segments change from the level corresponding to the site of injury.

Grade 1 Recovery.—Return of function in more than 2, but less than 4 segments.

Grade 2 Recovery.—More than 4 but less than 6 segments recovered functionally.

Grade 3 Recovery.—More than 6 segments or complete return of function.

RESULTS

1. *Severity of Injury.* As indicated previously, it was usually difficult to determine from reading the reports how complete or severe had been the injury to the spine initially. Although the operative data frequently were informative in cases of complete destruction of the cord, gross inspection of less severe injuries did not afford a reliable criterion for prognosis. Insufficient data were obtained regarding radiological examination for bony en-

croachment upon the spinal canal to help in this study. Thus, conclusions regarding the extent and irreversibility of damage to the intradural contents were reached mainly from the neurological findings described throughout the course of the patient. In the great majority of cases, complete, or nearly complete loss of function referable to the site of the lesion existed initially. For this reason it is evident in comparing the results of operative and non-operative treatment that laminectomized patients usually had more severe injuries than did those conservatively managed. In reviewing the available data on spinal manometrics, the above statement is supported by the fact that most of the surgically treated patients so tested had subarachnoid block, and most of those who were not operated upon had no block.

2. *Subarachnoid Studies.* Some information concerning the initial or early states of spinal fluid dynamics was available. Statements concerning the presence or absence of complete spinal subarachnoid block were obtained in 252 instances. Table 1 indicates that the results following both operative and non-operative treatment were significantly better when no

TABLE 1

Results following operative and non-operative treatment in 252 cases

Treatment	No. of Patients	No. Improved	Per Cent Improved
Block			
Laminectomy	100	12	12%
Non-laminectomy	25	0	0%
No Block			
Laminectomy	21	6	29%
Non-laminectomy	106	40	38%

block existed, than when complete obstruction was demonstrated. In an additional 9 patients partial block was said to be present, but these cases were not included in Table 1 because criteria for making a judgment of incomplete obstruction were found to be quite variable. When complete block was present, however, the figures definitely indicated that patients did better following laminectomy than when they were not operated upon.

Comment. Quite clearly, the patients with no block were less severely injured than were those who exhibited subarachnoid obstruction. When block did occur, however, laminectomy afforded some, if not great, benefit.

3. *Time of Treatment.* From Table 2 it can be seen that significant improvement occurred in 16 per cent of patients upon whom laminectomy was done and in 29 per cent of those who were not operated upon. It was somewhat surprising to find that the incidence of improvement was about the same in the patients who underwent surgery as long as 1 month after injury as it was in those explored earlier; and some benefit resulted to those operated upon up to a year later. Moreover, it did not appear on further evaluation that the degree of recovery was any more significant in the cases

of early, as opposed to later operation, at least in those patients explored up to 1 month after injury. About half of the 91 patients who improved after operation subsequently became ambulatory (47 cases or 8 per cent of those operated upon). In reviewing the records of these 47 patients it became evident that 25 had been operated upon more than 3 days after injury, and 3 more than a month after.

Comment. Again, the patients not operated upon appear to have been less severely injured than those who were treated surgically. Of the patients treated conservatively, about 3 in 10 showed some improvement and about 3 in 20 recovered or were rehabilitated to the point of ambulation. When laminectomy was necessary, 3 in 20 improved and 3 in 40 walked. Of importance in the surgically treated group seemed to be that the improvement

TABLE 2

Results following operative and non-operative treatment in total series of 858 cases

Time Interval Between Injury and Operation	Total Cases	No. of Patients Improved	Per Cent Improved	No. of Patients Ambulatory	Per Cent Ambulatory
With Laminectomy					
1–12 hrs.	97	15	16%	7	7%
12–24 hrs.	86	12	14%	5	6%
24–48 hrs.	36	10	28%	7	19%
48–72 hrs.	22	6	27%	3	14%
3 days–1 wk.	63	9	14%	6	9%
1 wk.–1 mo.	125	25	20%	16	13%
1 mo.–3 mos.	70	6	9%	1	1%
3 mos.–6 mos.	37	5	14%	1	3%
6 mos.–1 yr.	26	3	12%	1	4%
over 1 yr.	17	0	0%	0	0%
Total	579	91	Av. 16%	47	Av. 8%
Without Laminectomy	279	80	29%	44	16%

following exploration was as frequently obtained and apparently as extensive in patients operated upon from a week to a month after injury as in those who had an early operation. These results suggest that the acute paraplegic may profitably be treated with deliberation, not only to assess the need for intervention, but also to prepare him for it.

4. *Location of Injury.* The results of the follow-up in patients, according to the region of the intraspinal structures injured, is summarized in Table 3. Improvement in the patients not operated upon was more frequent than in those treated surgically, but the two series cannot be compared in this regard for reasons previously stated. The least improvement occurred when the upper thoracic cord was injured, and the greatest recovery followed lesions of the lumbar area. As lumbar injuries commonly resulted in injury to nerves, rather than to the spinal cord, an evaluation of recovery in these

TABLE 3

Results according to region of injury

Region of Injury	Total Cases	No. of Patients Improved	Per Cent Improved	No. of Patients Ambulatory	Per Cent Ambulatory
With Laminectomy					
Cervical	61	12	20%	5	8%
T1–6	115	10	9%	3	3%
T7–12	285	32	11%	19	7%
L	118	38	32%	20	17%
Total	579	92	Av. 16%	47	Av. 8%
Without Laminectomy					
Cervical	115	36	31%	13	11%
T1–6	32	3	9%	1	3%
T7–12	84	16	19%	8	10%
L	48	25	52%	22	46%
Total	279	80	Av. 29%	44	Av. 16%

two structures was made (Table 4). It was found that improvement occurred to a greater extent in nerves than in the cord. Moreover, as demonstrated before, patients with early operations did not do so well as those explored between the 2nd and 30th day after injury, if the injury involved the spinal cord. This differential in recovery was not apparent in patients with injuries to intraspinal nerves, both groups doing about equally well.

Comment. It was apparent in reviewing the cases, that recovery in patients with injuries to intraspinal nerves appeared most frequently within several months of injury, as it did in instances of trauma to the spinal cord. This observation suggested that the increased frequency of improvement

TABLE 4

Comparison of recovery in spinal cord and intraspinal nerves after lumbar injuries

Time Interval Between Injury And Operation	Injury to Spinal Cord			Injury to Intraspinal Nerves		
	No. of Cases	No. of Patients Improved	Per Cent Improved	No. of Cases	No. of Patients Improved	Per Cent Improved
With Laminectomy						
1–24 hrs.	113	8	7%	66	16	25%
1 day–1 mo.	145	26	18%	98	24	25%
1 mo.–1 yr.	93	8	9%	58	7	12%
	351	42	Av. 12%	222	47	Av. 21%
Without Laminectomy	194	47	24%	82	31	38%

among the lumbar injuries was not caused by regeneration but by return of function in nerve fibers. It further indicates that trauma to neural structures is tolerated better by the mobile and yielding cauda equina than by the bulkier and less displaceable spinal cord.

The figures on cervical injuries may give some indication regarding the contribution of neurological return of function to ultimate ambulation. Certainly, the completely tetraplegic patient would be totally incapable of ambulation, unless restitution of neural pathways occurred, while an individual with a lumbar lesion might ultimately walk even though his neurological status remained unchanged. Such recovery, as manifested both by neurological examination and ultimate ambulation, was higher for people with cervical injuries than for those with spinal lesions anywhere else but in the lumbar area. Realizing the appallingly handicapped plight of the tetraplegic patient, some slight gratification can be derived, perhaps, from

TABLE 5

Relative recovery of various modalities of function

Function	579 Cases With Laminectomy				279 Cases Without Laminectomy				858 Cases With and Without Laminectomy			
	Any Return	Per Cent	Great Return	Per Cent	Any Return	Per Cent	Great Return	Per Cent	Any Return	Per Cent	Great Return	Per Cent
All systems	92	16%			80	29%			172	20%		
Tactile	87	15%	39	7%	79	28%	60	22%	166	19%	99	11%
Pain and temperature	83	14%	16	3%	63	22%	17	6%	146	17%	33	4%
Motor	84	14%	9	2%	62	22%	15	5%	146	17%	24	3%
Bladder sensation	29	5%	26	5%	28	10%	25	10%	57	7%	51	6%
Bladder control	28	5%	24	5%	19	7%	15	5%	47	5%	39	5%

the fact that he has a 20–31 per cent chance of improving and an 8–11 per cent chance of walking.

The apparently more severe nature of the thoracic spinal injuries as compared to the cervical injuries is probably on the basis of the relative magnitude of forces required to produce fracture dislocations in these areas.

5. *Recovery of Different Modalities of Function.* The various modalities of spinal cord and nerve function differed in the tendency to recover functionally from injury. In all categories the intraspinal nerve lesions were followed by a greater frequency of recovery than the spinal cord lesions. Sensory functions recovered more frequently than motor functions, and autonomic functions (bladder) returned least of all. Table 5 illustrates the relative recovery rates among these various functional modalities. Tactile sensibility returned most frequently (19 per cent of all cases). Pain and temperature sensation as well as motor function returned next most frequently (17 per cent of all cases), while bladder sensation (7 per cent) and bladder motor control (5 per cent) showed significantly less tendency to recover. Analysis

of the modalities with regard to degree of recovery indicates that the tactile sensibility tended to improve to a marked degree much more frequently than any other function. Pain and temperature sense showed major recovery much less frequently, but more consistently than did skeletal motor function. Bladder motor and sensory function returned least frequently, but when they did return, it was to a close approximation of normal function, in most cases. Significantly it was noted that in no case did bladder sensation recover unless pain and temperature sense returned in the sacral segments. Review of Table 5 indicates again the tendency in the non-surgical cases toward much higher recovery rates in all modalities except bladder motor control.

Comment. The reasons for preservation of tactile sensibility (light touch, position and vibration senses) more frequently than other modalities are not apparent. In cord injuries one might suppose that the posterior funiculi are less susceptible to trauma in anterior compression (flexion) injuries because of the attachment of the denticulate ligaments.[7] Such a distinction could not explain the similar preservation of tactile senses in cauda equina lesions.

TABLE 6

Ambulation after laminectomy

Type of Lesion	Total Cases	Total No. Improved	No. of Patients Ambulatory			
			Minimal Aid*	Moderate Aid*	Marked Aid*	Total No. Ambulatory
Upper motor neuron	351	41	7	8	2	17
Cervical			5			
T1–6			1	2		
T7–12			1	6	2	
Lower motor neuron	222	48	6	3	18	27
T7–12			1		8	
L			5	3	10	
Upper and lower motor neuron	6	3			3	3
T7–12					2	
L					1	
Total	579	92	13	11	23	47

* Minimal aid—no support or 1 short leg brace only.
Moderate aid—canes or Canadian crutches with or without 1 short leg brace.
Marked aid—canes or crutches with 2 short leg braces or 1 long leg brace.

The observed relationship of bladder sensation to sacral pain and temperature innervation confirms the work of Nathan and Smith,[10] who found that pathways mediating bladder sensation course primarily in the anterolateral funiculi.

6. *Ambulant Patients.* Of great interest to the patients and to those interested in them is the prognosis as regards ambulation. For this reason a

study was made of the cases to evaluate the degree and frequency of return of this general function. In Table 2 was a summary of all ambulatory patients. These data are broken down in Tables 6 and 7 to indicate the relative degree of ambulation achieved by the patients. We did not consider patients to be "ambulatory" if they required bilateral long leg braces. The patients listed walked usefully at least some of the time, or were at least neurologically capable of doing so.

Of 579 laminectomized patients, 13 walked with no aid or with one drop-foot brace. Actually 7, or 1.2 per cent, walked without aid. Eleven patients required moderate aid, and 23 walked with marked assistance as described in Table 6.

Of 279 patients without laminectomy, 20 walked with minimal aid and

TABLE 7

Ambulation without laminectomy

Type of Lesion	Total Cases	Total No. Patients Improved	No. of Patients Ambulatory			
			Minimal Aid	Moderate Aid	Marked Aid	Total No. Ambulatory
Upper motor neuron	194	47	11	7		18
Cervical			10	3		
T1–6			1			
T7–12				3		
L				1		
Lower motor neuron	82	31	9	10	6	25
T7–12			2	2	1	
L			7	8	5	
Upper and lower motor neuron	3	2			1	1
T7–12						
L					1	
Total	279	80	20	17	7	44

16, or 6 per cent, walked with no aid at all. Seventeen required moderate aid and 7 marked assistance. Thus, again, was the more benign lesion in this group demonstrated.

Final Comment. When confronted with the acute traumatic paraplegic the attending physician is faced with the problem of deciding upon management that will afford the maximum opportunity for spinal cord or nerve recovery. At best, the chances are small for improvement. However, the patient will not be content unless every procedure that can possibly improve the prognosis is performed. After complete neurological examination, lumbar puncture and x-ray studies, the physician must conclude whether doubt does or does not exist regarding the complete anatomical destruction of the spinal cord or nerves at the site of the injury. If there is no doubt that the neural structures are completely destroyed, then laminectomy is not

indicated. If such doubt exists, as it does in most cases, then the decision of when or whether to explore depends upon the general condition of the patient, his neurological course, the x-ray findings and the spinal fluid manometrics. If his condition is such that operation would seriously imperil his survival then it should be deferred until a more opportune time. Empirically we agree with prevalent opinion that progressive neurological decline indicates laminectomy, and also that progressive recovery contraindicates it. In cases of non-progressive severe neurological deficit when doubt exists regarding complete anatomical destruction of the neural structures affected, and when the patient's general condition is good, the decision about laminectomy rests on the evaluation of the spinal x-ray findings and the spinal fluid manometrics. We have not fully evaluated the relationship of x-ray appearance of the spine to prognosis in this study, but feel like Brown[1] that it is not constantly reliable. Our data on spinal fluid block indicate that the presence of block seriously decreases the prognosis, and that if not operated upon, the patient's chance of neurological recovery is minimal. It has not been possible here to assess the effect of disappearance of spinal fluid block, upon return of function, as edema subsides under conservative treatment or as skeletal traction reduces the dislocation. However, the results of this review indicate that 1 or 2 days' waiting will not seriously imperil the prognosis, and in fact may even improve it.

In the event of paraplegia in the absence of spinal fluid block, the problem is quite different. The prognosis seems considerably better. The absence of block is usually associated with a lesser degree of vertebral displacement. Therefore, we believe that the absence of block in general indicates that the neural structures are not being seriously compressed, that maximum destruction has already occurred, and that nothing is to be gained by laminectomy. We have not specifically scrutinized the cases of paraplegic patients who demonstrate initial preservation of tactile sensibility and who, according to Schneider, benefit from laminectomy even though no block exists. It would seem that these cases represent an even less severe type of injury to the cord than the cases in which there is complete loss of neurological function and that, therefore, the prognosis should be even better among such patients without manometric block. For these reasons, we are not convinced of the value of laminectomy in such instances, an operation which still must be considered a major surgical procedure, despite the fact that it caused no fatality in our series.

SUMMARY

1. A series of 858 cases of spinal cord injury were reviewed. In 579 of these laminectomy was performed at various intervals after injury. In 279 cases exploration was not made. The neurological recovery among both groups was evaluated.

2. Of the laminectomized patients, 16 per cent were significantly improved postoperatively, and 8 per cent were ambulatory, 1.2 per cent with-

out aids. Of the patients not operated upon, 29 per cent improved neurologically, and 16 per cent were ambulatory, 6 per cent without aids. The laminectomized patients were felt to represent more severe injuries than the non-laminectomized patients for reasons outlined.

3. In both categories, the intraspinal nerve lesions recovered more frequently than the spinal cord lesions. This was thought to represent greater tolerance to trauma among the nerves of the cauda equina, rather than regeneration, for reasons stated.

4. In all patients it was found that systems conducting tactile sensibility recovered most frequently. Pain and temperature sensation and motor function returned next most frequently, and bladder sensation and motor control recovered least frequently of all functions. Possible factors are discussed.

5. Among the laminectomized patients the most favorable interval between injury and operation was from 24 hours to 1 month. Those operated upon earlier seemed less likely to improve if they had spinal cord lesions. Lesser rates of improvement were observed in all types of lesions operated upon more than 1 month after injury.

6. Spinal subarachnoid block was an important factor influencing prognosis, affecting it quite adversely. Patients with complete block who were not operated upon did not improve.

7. Gross observation of the cord at surgery did not afford a reliable indication of prognosis unless the cord was obviously transected. Many patients described as having "normal-appearing cord" did not improve, and many with subtotal lesions did improve.

8. Indications for and against surgery are discussed. (a) The presence of subarachnoid block is a strong indication for surgery if x-ray evidence of bony injury leaves any doubt as to completeness of the injury. (b) Absence of subarachnoid block supports the institution of non-surgical treatment initially. The anterior cord compression syndrome is discussed in the light of the above-mentioned indications and experience. (c) Laminectomy delayed as long as 1 year after injury may rarely afford some improvement in cases of complete block, but if delayed longer it probably affords only psychological benefit to the patient.

The authors wish to acknowledge their appreciation to Dr. Ernest Bors and Dr. John French for their many suggestions.

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CASE REPORTS AND TECHNICAL NOTES

OVERT DIABETES INSIPIDUS INDUCED BY CORTICOTROPHIN FOLLOWING EXCISION OF CHROMOPHOBE ADENOMA

CASE REPORT

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(Received for publication June 7, 1955)

Corticotrophin (ACTH) and cortisone have proved valuable in the preoperative and postoperative care of patients with hypophysial and parahypophysial neoplasms, many of whom have varying degrees of hypopituitarism.[3,9] Raaf *et al.*[18] described the use of corticotrophin without complications in 18 consecutive patients with pituitary or parasellar tumors.

The following case is reported to illustrate one of the possible adverse side effects of corticotrophin therapy in patients with pituitary tumors.

CASE REPORT

A 36-year-old male longshoreman was admitted to the University of California Hospital on Feb. 20, 1954, because of headaches and loss of libido of 6 months' duration, and progressive visual impairment present for 1 month.

Examination. The patient was moderately obese. Genitalia were considered normal in size and firm by an endocrinological consultant. Complete bitemporal hemianopia was present, but there were no other neurological abnormalities.

Roentgenograms of the skull disclosed an enlarged sella turcica.

Complete blood count and urinalysis were within normal limits. Specific gravity of urine was 1.022. The basal metabolic rate was minus 6 per cent. Fasting blood sugar was 97 mg. per cent (Somogyi). The eosinophil count fell from 100/mm.^{3} to 50 mm.^{3} 4 hours following the subcutaneous injection of 0.5 cc. of 1:1000 epinephrine. The serum sodium concentration was 131 mEq./l., potassium 3.9, chlorides 102, and CO_2 28.7. The 24-hour urinary excretion of 17-ketosteroids was 4.0 mg. (total neutral fraction, method of Drekter *et al.*[5]).

Operation. On Feb. 25, 1954, under general anesthesia, a right frontotemporal craniotomy was performed with subtotal removal of a pituitary chromophobe adenoma.

Postoperative Course. The visual fields rapidly returned to normal. The serum sodium concentration dropped to 116 mEq./l. on the 2nd day after operation, but was up to the preoperative level on the 6th day. The lowest serum chloride concentration recorded was 91 mEq./l. on the 4th postoperative day. Blood eosinophil levels dropped as expected during the immediate postoperative period (Fig. 1).

On the 9th postoperative day, at the suggestion of an endocrinological consultant, treatment with corticotrophin gel, 40 units per day in 2 divided doses by intramuscular injection, was begun as a prophylactic measure against possible adrenal insufficiency. On the following day, fluid intake increased to 4255 cc. and rose within the next 24 hours to 8265 cc., while urine output was 4125 cc. The daily intakes and outputs are charted in Fig. 1. Maximum 24-hour fluid intake was 9880 cc.; maximum urine output was 9250 cc. on March 12 (15th postoperative day). The dosage of corticotrophin was decreased during the next 5 days and then was discontinued. The excessive fluid exchange subsided concomitant with the gradual discontinuance of corticotrophin.

The patient received a course of radiation therapy to the region of the sella turcica. He subsequently returned to work. When examined 8 months after operation, the visual fields were normal. The patient reported that his fluid intake and output had not been excessive since the corticotrophin was discontinued.

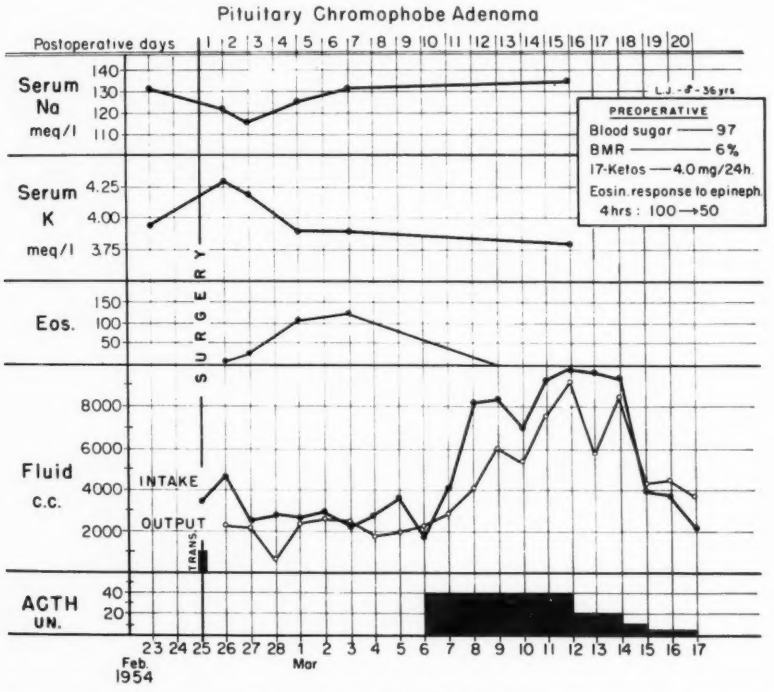

FIG. 1. Chart of pertinent data in case reported. From above downward—serum sodium concentration in milliequivalents per liter (mEq./l); serum potassium concentration (mEq./l.); blood eosinophils; fluid intake (solid circles) and fluid output (empty circles); and dose of corticotrophin.

DISCUSSION

Intact anterior hypophysial function is thought to be essential to the maintenance of overt diabetes insipidus, the effect probably being mediated by the trophic hormones of the anterior lobe.[7] Several authors have pointed out that the adrenal cortex and posterior pituitary are antagonistic in their action on water metabolism.[1,7,21] A syndrome similar to diabetes insipidus has been produced in normal dogs by administration of desoxycorticosterone acetate[15,16,19] and cortisone.[17,22,23]

In rats subjected to hypophysectomy, the transient diabetes insipidus which lasts for about 1 week after the procedure can be restored by the administration of desoxycorticosterone acetate.[4] Selye and Bassett[20] also found that desoxycorticosterone caused an increase in fluid intake and output in normal and hypophysectomized rats, but particularly in the latter.

In a series of 18 hypophysectomized patients who received either cortisone or corticotrophin, diabetes insipidus developed in 9 postoperatively.[25] In these in-

stances, it was subsequently demonstrated that the hypophysectomy was incomplete, whereas autopsies in 5 of the 9 cases in which diabetes insipidus did not develop confirmed complete removal of the pituitary. Luft[12] reported that following hypophysectomy in the human, cortisone induced polyuria of low specific gravity and polydipsia, which subsided if cortisone was withdrawn. However, the polyuria tended to disappear in 4 to 5 months, even if cortisone was continued.

Desoxycorticosterone caused a three-fold increase in the urinary volume of a patient with long-standing panhypopituitarism caused by a craniopharyngioma.[14] Lloyd[11] mentioned a patient with diabetes insipidus, the severity of which varied with the urinary corticosteroid excretion. When the corticosteroid output was 1.25 mg./24 hours, the patient required 20 units of pitressin tannate daily to control the diabetes insipidus. Two years later, the corticosteroid output was 0.275 mg./24 hours and the pitressin requirement was only 1.6 units daily. In Case I of Ingraham *et al.*[9] the patient received corticotrophin and thyroid substance before and after craniotomy for partial removal of a craniopharyngioma. Transient diabetes insipidus developed on the 4th postoperative day.

Engstrom and Liebman[6] studied a patient with supposed co-existing diabetes insipidus and anterior pituitary insufficiency. The presence of diabetes insipidus appears doubtful, however, since from the data given in their charts, the daily fluid intake and output were hardly excessive during periods when no pitressin was administered.

In the patient reported by Leaf *et al.*,[10] polyuria and polydipsia subsided as anterior pituitary failure developed. Polyuria was not re-established by administration of corticotrophin, cortisone, thyroxin, desoxycorticosterone or growth hormone when the excretory solute load was kept constant.

The evidence of preoperative hypopituitarism in the patient reported here consisted of a history of loss of libido, the urinary 17-ketosteroid excretion of 4 mg./24 hours, and the serum sodium concentration of 131 mEq./l. In spite of the fall in serum sodium concentration to 116 mEq./l. on the 2nd postoperative day, there was no clinical evidence of an Addisonian crisis, and the serum sodium concentration rose promptly without specific therapy. One day after corticotrophin therapy was begun, fluid intake and output increased. This excessive fluid exchange progressed during the next several days to a level consistent with true diabetes insipidus, then subsided as the corticotrophin was gradually discontinued.

Unfortunately, the response of water diuresis to hypertonic saline[2,8] was not tested in our patient.

With the increased use of corticotrophin in patients undergoing surgical excision of pituitary tumors, experiences similar to that described in the present report will probably be encountered. In the present case, there were no apparent deleterious effects since a negative water balance did not develop. However, the onset of true diabetes insipidus in the unconscious (and incontinent) postoperative patient, if unrecognized, may rapidly cause a severe dehydration and hyperosmolarity of extracellular fluids. Welt *et al.*[24] emphasized this danger, and reported a patient whose serum sodium concentration reached 171 mEq./l. on the 1st postoperative day, because of the acute onset of diabetes insipidus.

SUMMARY

The administration of corticotrophin (ACTH) to a patient following subtotal removal of an hypophysial chromophobe adenoma caused a marked increase in fluid intake and output. This excessive fluid exchange subsided simultaneously with the

discontinuance of corticotrophin. Pertinent previous studies bearing on the problem are discussed.

Doctors Edwin B. Boldrey and Gilbert S. Gordon gave valuable advice and criticism in the preparation of this report.

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UNILATERAL THORACIC HYPERHIDROSIS CAUSED BY OSTEOMA OF THE TENTH DORSAL VERTEBRA

CASE REPORT

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(Received for publication August 10, 1955)

Because of its diagnostic, physiological, and therapeutic interest an unusual case of ascending unilateral hyperhidrosis is reported in which plurisegmental disturbance of sweating was cured by removal of an osteoblastic osteoma and extradural rhizotomy at the level of the 10th thoracic vertebra.

CASE REPORT

R.K., a young girl of 15½ years, was referred to the Neurological Institute of New York by Dr. William Lazarus of Brooklyn on Nov. 19, 1953. Her chief complaint was progressively severe sweating over the left side of the chest and axilla of 5 months' duration. Hyperhidrosis had begun in the left 10th dorsal dermatome whence it had successively involved all the thoracic dermatomes from D10–D2 and D10–D12 inclusive (Fig. 2A). The perspiration had become so profuse and embarrassing that she gave up school and all social activities. It usually drenched her clothing on the affected side within 5–10 minutes after a change of garments and was always accentuated by excitement or emotional tension.

Less disturbing associated symptoms were mild aching pains just to the left of the 10th dorsal vertebra of 3 years' duration, and an intermittent "pins and needles" sensation in the same area, progressive left thoracic scoliosis, and occasional precordial sticking pains of 5 months' duration. There were no other neurological symptoms.

The menarche began at 12½ years of age and at this time hirsutism of the face occurred, for which occasional treatments by electrolysis had been done ever since. The menstrual periods had been regular and normal, while metabolic and endocrine studies revealed no gross dysfunction.

There was no history of significant injury other than dislocation of the left knee while

FIG. 1. Roentgenogram showing rounded translucent area involving lamina and base of transverse and spinous processes of D10 vertebra.

playing volleyball, and there had been no serious illnesses, infections or systemic disease. The birth history and family history were noncontributory.

Examination. The patient was an alert young girl whose dress and skin were wet with sweat from the left axilla to the level of D12 inclusive. The left palm was slightly more moist than the right but there was no Horner's syndrome. Moderate scoliosis of the thoracic spine to the left was present and some tenderness was elicited, together with minimal hyperpathia, just to the left of the 10th dorsal spinous process. Hirsutism over the face and chin, and to a lesser extent over the trunk and extremities, was also noted. Blood pressure was normal.

Laboratory Data. Blood count and urinalysis were normal. Erythrocyte sedimentation rate was 20 min. per hour. CSF pressure and manometric tests were normal; the fluid was

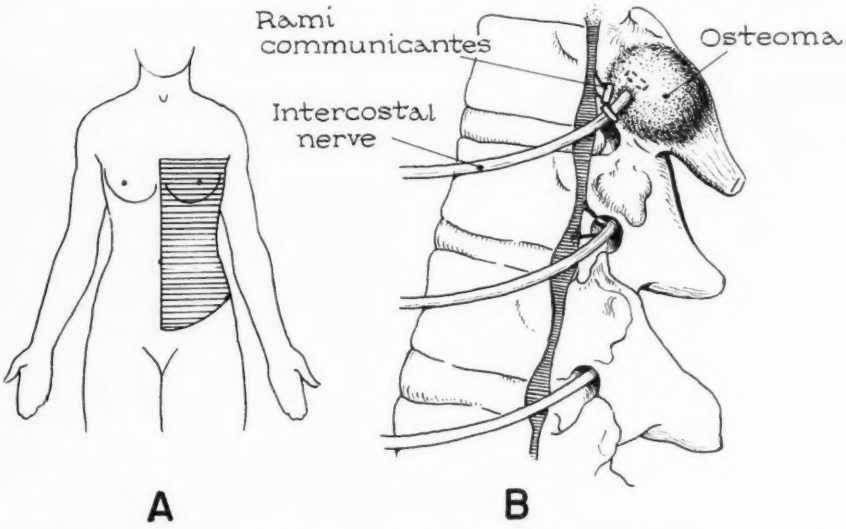

FIG. 2. (A) Hyperhidrosis involved shaded area from left axilla to D12 dermatome inclusive, anteriorly and posteriorly. Complete relief of hyperhidrosis and pain with marked improvement of scoliosis followed removal of tumor and resection of nerve and nerve roots, indicated in B. (B) Diagram showing site of osteoma with encroachment upon intervertebral foramen at D10-11, left. Nerve roots were divided extradurally distal to silver clip (broken lines); intercostal nerve and rami communicantes were divided proximal to silver clips (solid lines).

crystal clear and contained 1 wbc.; total protein was 38 mg. per cent; Kolmer test was negative.

Roentgenograms of the chest were normal. Films of the spine showed mild thoracic scoliosis to the left of about 8 degrees. A radiolucent shadow, 1.8 cm. in diameter, overlay the left side of the body of the 10th dorsal vertebra (Fig. 1). Laminograms revealed that this was a destructive lesion involving the left lamina of D10 and the base of the left D10 transverse and spinous processes. Myelograms were normal, save for very slight extradural indentation at D10–11 on the left.

Operation. On Nov. 27, 1953 hemilaminectomy, D10–11, left, was performed, with removal of unusually vascular, softened and somewhat thickened bone comprising the left lamina and adjacent portions of the transverse and spinous processes of the 10th dorsal vertebra. The intervertebral foramen at D10–11 was partly occluded by the abnormal bone (Fig. 2B), but there was no other evidence of a pathological process, intraspinally, or extraspinally in the paravertebral gutter.

All grossly pathological bone having been removed, the subjacent nerve was resected from the dura mater to a distance of 2.5 cm. laterally. This included the anterior and dorsal roots, the posterior root ganglion within the dural sleeve, and the rami communicantes (Fig. 2B). The adjacent sympathetic ganglion and chain were not disturbed.

Microscopic Diagnosis. (a) Bone: osteoblastic osteoma, benign (Fig. 3). (b) Nerve: normal nerve elements.

Postoperative Course. There were no complications and the patient has not suffered any further sweating abnormalities or pain during the 1½-year follow-up period. The scoliosis has also virtually disappeared and recent roentgenological studies have revealed no recurrence of the pathological process.

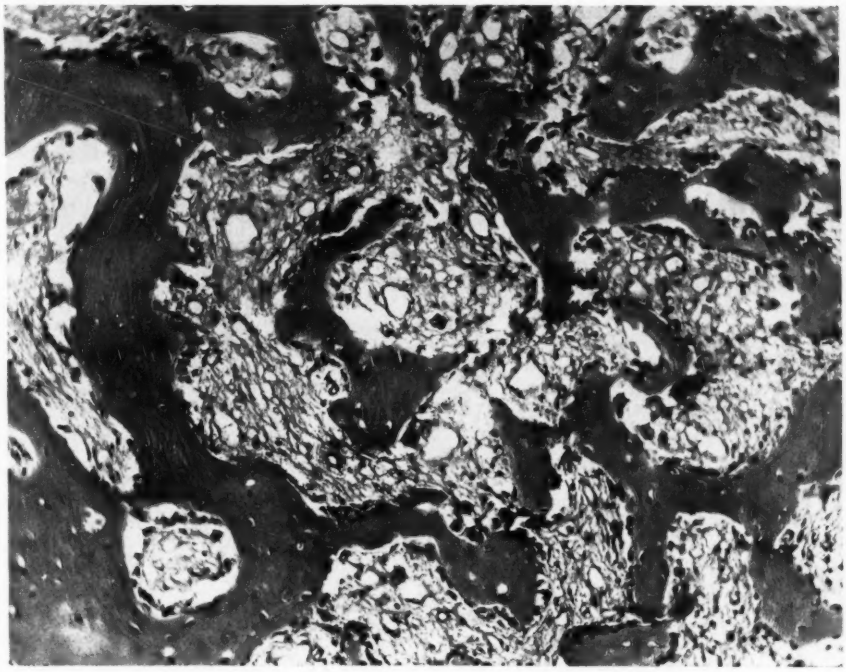

FIG. 3. Microscopic appearance of tumor, an osteoblastic osteoma.

DISCUSSION

(a) *Differential Diagnosis.* Unilateral hyperhidrosis is rare, but has been described in isolated instances over dermatomes affected by an intramedullary glioma[8] or syringomyelic cavity[1,3] of the spinal cord, and below the level of traumatic hemisection of the cord.[4] It has also occurred over one side of the face during the onset of focal convulsive seizures secondary to an oligodendroglioma of the postero-inferior portion of the frontal lobe.[2] Tumors involving the sympathetic chain or ganglia may presumably result in hyperhidrosis during a stage of irritation; that is, before a pathological sympathectomy by tumor compression or invasion has occurred, although the author has not seen such a phenomenon.

In this case the differential diagnosis on clinical grounds lay between intrinsic

disease of the spinal cord such as a glioma or syrinx, and an extradural or paravertebral tumor such as a dumb-bell type of ganglioneuroma, neuroblastoma, or neurofibroma. The absence of spinal cord signs and the subsequent normal cerebrospinal fluid and myelographic findings ruled out intrinsic spinal cord disease. The clinical evidence of local D10 nerve or nerve root irritation plus the subsequent x-ray findings of a bone lesion at D10 pointed to an extradural process, presumably neoplastic because of the progressive symptomatology. The 3-year history of local pain militated against a malignancy. The possibility of nerve compression by scoliosis was tentatively entertained but discarded because the scoliosis did not seem sufficiently advanced. X-ray findings then disclosed the presence of a focal lesion characterized by a circular defect (Fig. 1), some irregularity and narrowing of the D10–11 intervertebral foramen, thinning of the adjacent pedicle, and scoliosis with the concavity to the left. It was felt that an extraspinal tumor mass might also be present to account for the clinical findings, and in particular, the hyperhidrosis. For this reason a neurofibroma or ganglioneuroma of dumb-bell type, mostly paravertebral, was strongly considered, with secondary pressure effects accounting for the vertebral changes, although a primary bone tumor was of course also considered. In retrospect, the character of the x-ray findings should have led to a diagnosis of primary bone tumor.

(b) *Physiology.* Laboratory[7] and clinical experience[5,6] has clearly established the following well known features of sympathetic innervation: (i) that autonomic anterior root fibers activate the sweating mechanism; (ii) that the 1st thoracic anterior root carries few if any fibers concerned with sweating compared with the 2nd thoracic; (iii) that section of a single or even several anterior roots (D3–9 inclusive, for example) may create no sweating loss; (iv) that electrical stimulation of a single anterior root generally results in widespread sympathetic effects over the distribution of at least five or six sympathetic ganglia; and (v) that there is a dermatomic overlap of cutaneous sympathetic innervation just as there is for sensory innervation of the skin.

All these criteria fitted the circumstances in the present case and proved useful in guiding the operative procedure. On these physiological grounds, for example, it was not deemed necessary to remove any sympathetic ganglia because the abnormal sympathetic activation seemed purely a phenomenon of focal irritation of the D10 anterior root fibers by the tumor and/or the constricted intervertebral foramen. It was presumed that abnormal activation of the D10 sympathetic ganglion in turn had served progressively to activate the rest of the ipsilateral thoracic sympathetic chain. The postoperative course indicated that this premise was correct.

It is of interest to note that sensory and motor irritative effects were also apparent, as indicated by the intermittent focal pain and paresthesias, and by the progressive scoliosis, respectively. The scoliosis, it was felt, could well be attributed to motor root activation resulting in mild chronic spasm of the left paravertebral muscles sufficient to cause the spinal distortion by a bow-string action.

(c) *Therapeutic.* In this case removal of the pathological bone and foraminotomy alone would probably have sufficed to relieve the partial nerve root and nerve compression leading to the described symptomatology.

However, the nerve roots and nerve were sectioned because of the possibility that their chronic compression might have induced permanent intraneural changes that might perpetuate preoperative symptomatology. No x-ray therapy was given be-

cause all the pathologists who examined the sections of this tumor agreed that it was benign.

SUMMARY

1. Lasting relief of unilateral hyperhidrosis from D2-D12 inclusive has been accomplished by removal of a small osteoblastic osteoma involving the 10th thoracic vertebra. Resection of a segment of the nerve and the extradural portion of its nerve roots at D10–11 on the left apparently contributed to the enduring relief of the hyperhidrosis, pain, paresthesias and scoliosis described in this case.

2. The differential diagnosis is briefly considered.

3. Correction of abnormal neural activation at one level (D10) restored sympathetic sweating activity to normal at other levels (D2 through D12 inclusive). Reasons for this phenomenon are discussed in the light of established laboratory and clinical data.

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DEPRESSED COMMINUTED FRACTURE OF A PLASTIC CRANIOPLASTY

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A depressed comminuted fracture of a large plastic (methyl methacrylate resin) cranioplasty and lacerated dura mater following a minor head injury is an unusual enough complication to warrant a brief report. The advantages of this plastic material over other materials for repair of cranial defects are the ease of perfect molding, the low cost, and the x-ray translucency, as Spence[1] has reported. However, its brittleness makes one reluctant to use it for large bony defects, as is brought out in the following case report.

CASE REPORT

W.H., a 27-year-old white male, was first admitted to the Neurosurgical Service of John Sealy Hospital in February, 1953, with signs and symptoms referable to a left cerebral neoplasm. A left parietal craniotomy was performed on Feb. 24, 1953 with subtotal removal of a grade III glioma. On the 2nd postoperative day the wound was reopened and an intracerebral hematoma was removed. The patient improved but soon a subgaleal and intracerebral abscess developed. The bone flap and abscess were removed on Mar. 3, 1953.

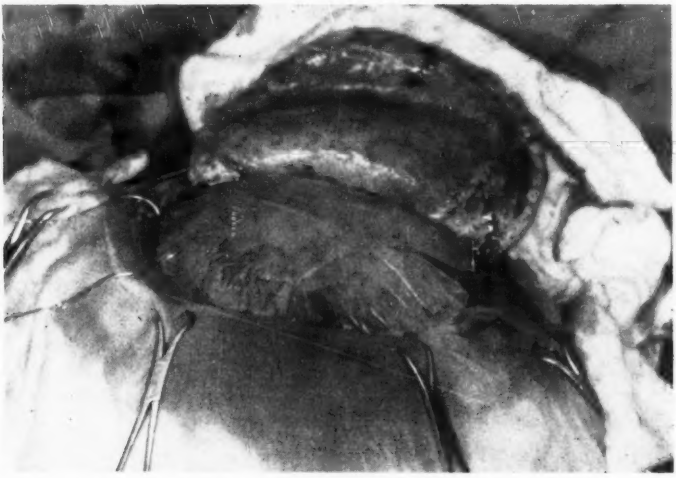

FIG. 1. Operative view of plastic plate. Arrow points to depressed fragments and lacerated dura mater.

His postoperative course was otherwise uneventful, and he was discharged on Apr. 1, 1953. During the following year, the patient did rather well except for complaints of dizziness

and headache, particularly when he bent down or got up, and his cranial defect showed marked depression.

Since the patient had done so well postoperatively for 18 months, as far as his tumor was concerned, he was readmitted to the hospital for repair of his cranial defect.

On Aug. 2, 1954, a plastic (methyl methacrylate) cranioplasty was molded at surgery and fixed into the bony defect by wire sutures.

After this procedure he no longer complained of headache and dizziness and was able to do his daily chores around the farm. However, on Aug. 21, 1954 he was involved in an automobile accident in which he struck his head on the top of the car. He was not rendered unconscious nor was there a laceration of the scalp. Immediately following this he complained of headache and could palpate some fluid between the scalp and the plate. There was some progression of his neurological deficit in that his aphasia was slightly more evident as was the sensory ataxia of his right arm. Therefore, he was again admitted to the hospital.

On Aug. 31, 1954, operation was performed. It was seen that the plastic plate was fractured in three places (Fig. 1). One fragment was depressed and had lacerated the dura mater in two places and cerebrospinal fluid had leaked outside the dura mater into the subgaleal area. Tissue that was adjacent to the plastic plate was excised and sent for microscopic studies to Dr. Kenneth M. Earle, our neuropathologist, who reported that it was composed of dense fibrous and cartilaginous connective tissue with focal collections of chronic inflammatory cells which consisted mostly of small lymphocytes. In the connective tissue there were scattered foci of foreign body giant cells containing foreign matter of double refractile character when exposed to Polaroid light. Therefore, the plastic plate was removed and a tantalum cranioplasty was done.

Since then the aphasia and sensory ataxia of the right arm have improved to the pre-accident level. The patient has returned to his chicken farm and is now working full time at this last report of August, 1955.

DISCUSSION

We caution against the use of such plastic material to cover large cranial defects as shown in this case, in which it fractured in response to a minor injury. This caused the depressed fragments of the plate to lacerate the dura mater. Had a tantalum plate been used originally, this situation would not have occurred and at the most there would have been only a dent. Therefore, plastic plates such as these probably are better used to cover small cranial defects and should not be used to fill in large ones.

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CLASSIFICATION OF NEUROSURGICAL CASES AND OPERATIONS*

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(Received for publication August 26, 1955)

The office registration of cases and operative procedures in the practice of neurological surgery presents the primary problems of (a) orderly recording, and (b) accessibility of statistics for reference work.

In meeting these problems, the basic requirement is a comprehensive outline of classification, and the adjunct is an efficient structure for filing. This combination provides organization of clinical material for review, statistical report or scientific paper.

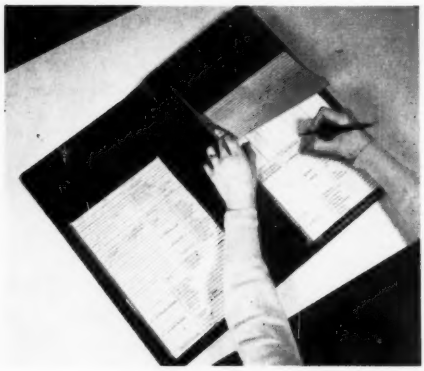

FIG. 1. Visible card file.

Major filing systems[1,2] used in hospital record libraries have been found too complex for the ordinary office register. The terminology is diversified to the point of redundancy. Tables 1 and 2 present outlines for classification of cases and operations respectively—designed according to incidence and relation. Revisions have been made in the course of practical application.

The outline has been fitted to a standard visible card file of the book-fold type as shown in Fig. 1. There is adequate room for the outline of cases and operations in the same file, as will be discussed below. This file is simple in maintenance and its portability is a distinct advantage in the office or for conference work.

The subdivisions and headings made on the cards for the cases and operations are illustrated in Figs. 2 and 3.

DISCUSSION

The structure of the outline has been developed to include general disorders seen in the practice of neurological surgery, with subheadings according to frequency of types, and a miscellaneous division in each general group for inclusion of the less frequent types. A number of relatively infrequent and dissociated disorders would be grouped under Item XIV. Miscellaneous (Table 1).

There are two types of cards used in the file (Figs. 2 and 3). The headings of the case cards indicate the patient's name, hospital unit number, admission and discharge dates, as well as operation, general data and follow-up information. In a group practice or partnership, initials or color signals may be used to indicate the

* Exhibit at the Harvey Cushing Society meeting, Quebec City, May 1955.

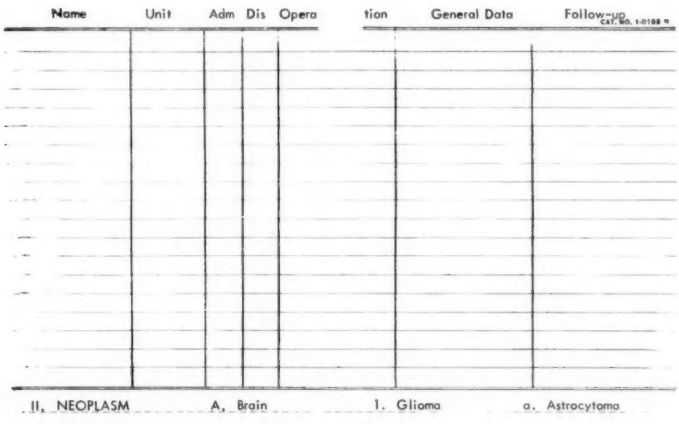

FIG. 2. Case card.

physician or hospital concerned. The space reserved for General Data may include such information as the type of accident involved in the cases of trauma, or other pertinent points in history or in hospital course. Space is provided for follow-up of the cases. Death may be recorded by the letter "D," followed by the date, and the letter "P," if postmortem examination is obtained. There is a wide range of flexibility in recording key factors.

The bookfold file (Fig. 1) contains 102 pockets, providing ample space for the outlines of cases and operations in one file. Each card contains 19 lines on either side and each pocket has room for storing at least 5 cards. Cases of relatively high frequency are assigned more than one pocket in the file. Overflow cards are filed in a separate container.

For cross reference to the bookfold file, a simple drawer file (containing 3×5

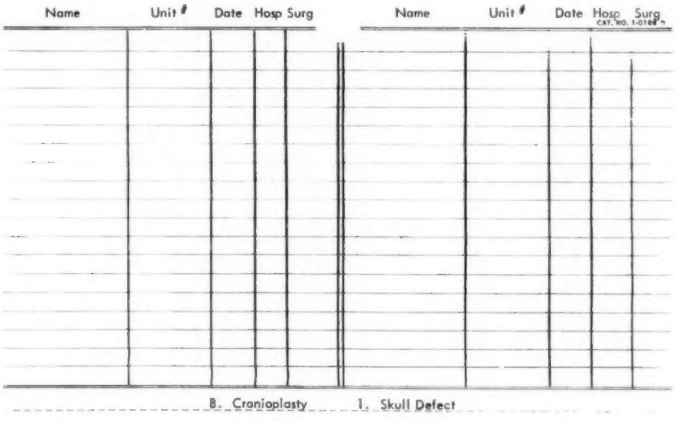

FIG. 3. Operation card.

TABLE 1

Cases

I. TRAUMA
- A. Head
  - 1. Fracture of skull
  - 2. Concussion cerebral
  - 3. Intracranial hematoma
  - 4. Post-concussive syndrome
- B. Spine
  - 1. Fracture without cord injury
  - 2. Spinal cord injury
  - 3. Intervertebral disc
    - a. Cervical-thoracic
    - b. Lumbar
    - c. Disc suspect
  - 4. Back Strain
- C. Peripheral nerve
- D. Miscellaneous

II. NEOPLASM
- A. Brain
  - 1. Glioma
    - a. Astrocytoma
    - b. Ependymoma
    - c. Glioblastoma
    - d. Medulloblastoma
    - e. Miscellaneous
  - 2. Mesodermal tumors
    - a. Meningioma
    - b. Neuroma
    - c. Angioma
    - d. Miscellaneous
  - 3. Pituitary tumor
    - a. Chromophobe adenoma
    - b. Chromophil adenoma
    - c. Craniopharyngioma
    - d. Miscellaneous
  - 4. Congenital tumors
    - a. Dermoid
    - b. Miscellaneous
  - 5. Metastatic tumor
- B. Spinal
  - 1. Intramedullary
  - 2. Extramedullary
  - 3. Metastatic
- C. Brain tumor suspect
- D. Spinal tumor suspect
- E. Miscellaneous

III. CONGENITAL DISORDERS
- A. Spina bifida
- B. Cranium bifida
- C. Craniosynostosis
- D. Dermal sinus
- E. Miscellaneous

IV. VASCULAR DISORDERS
- A. Aneurysm
  - 1. Arterial
  - 2. Arterio-venous
- B. Hemorrhage
- C. Thrombosis
- D. Arteriosclerosis
- E. Miscellaneous

V. INFECTIOUS DISORDERS
- A. Abscess
- B. Osteomyelitis
- C. Meningitis
- D. Encephalitis
- E. Miscellaneous

VI. CONVULSIVE DISORDERS
- A. Post-traumatic
- B. Post-infectious
- C. Cause undetermined
- D. Miscellaneous

VII. PAIN DISORDERS
- A. Tic douloureux
- B. Atypical facial neuralgia
- C. Intractable pain with malignancy
- D. Miscellaneous

VIII. PRIMARY NEUROLOGICAL DISORDERS
- A. Multiple sclerosis
- B. Amyotrophic lateral sclerosis
- C. Miscellaneous

IX. HYPERKINETIC DISORDERS
- A. Parkinsonism
- B. Miscellaneous

X. PSYCHIC DISORDERS
- A. Psychoneurosis
- B. Psychosis, organic
- C. Schizophrenia
- D. Miscellaneous

XI. BONE AND JOINT DISORDERS
- A. Arthritis
- B. Miscellaneous

XII. HYDROCEPHALUS
- A. Communicating
- B. Non-communicating

XIII. ARACHNOIDITIS

XIV. MISCELLANEOUS

cards) is maintained. The cards are arranged alphabetically and each gives the patient's name, address, telephone number, next of kin and similar data, as well as the outline number referring to location in the bookfold file. The same type of cross-reference file could be maintained with the office ledger sheets. Thus any patient may be traced by name or diagnosis.

TABLE 2

Operations

I. MAJOR OPERATIONS
- A. Craniotomy
  - 1. Brain tumor
  - 2. Aneurysm
  - 3. Tic douloureux
  - 4. Hematoma
  - 5. Depressed fracture
  - 6. Miscellaneous
- B. Cranioplasty
  - 1. Skull defect
  - 2. Craniosynostosis
- C. Laminectomy
  - 1. Disc
    - a. Cervical-thoracic
    - b. Lumbar
    - c. Exploration
  - 2. Tumor
  - 3. Cordotomy
  - 4. Miscellaneous
- D. Miscellaneous

II. MINOR OPERATIONS
- A. Trephine of skull (Expl.)
- B. Alcohol injections
- C. Miscellaneous

III. DIAGNOSTIC OPERATIONS
- A. Ventriculogram
- B. Pneumoencephalogram
- C. Arteriogram
- D. Myelogram
- E. Miscellaneous

The outline of classification may be used in filing other items such as photographs, pathological specimens or slides, so that the general reference material becomes coordinated.

The everyday practical features of this system are stressed in helping the clinician who is periodically searching for a previous case or meeting the recurrent questions of residents concerning incidence, surgery, survival and other factors.

SUMMARY

An outline is presented for classification of neurosurgical cases and operations, adapted to a visible card file.*

REFERENCES

1. American Medical Association. Standard nomenclature of disease and standard nomenclature of operations. E. P. Jordan, Ed. *Philadelphia: Blakiston Co.*, 1950, xv, 1022 pp.
2. World Health Organization. Manual of the international statistical classification of diseases, injuries, and causes of death. *Geneva: World Health Organization*, 1948–49, 6th rev., 2 vols.

* File and cards may be ordered from Scrantom's Book and Stationery Co., Inc., 334 Main Street East, Rochester 4, New York.

A METHOD OF CONVERTING AN OPERATING TABLE INTO A REFRIGERATING TROUGH

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(Received for publication September 26, 1955)

By means of suitable attachments, an operating table may be converted into a refrigerating trough thus avoiding the inconvenience of transferring the hypothermic patient from the cold bath to the operating table. The necessary attachments are a

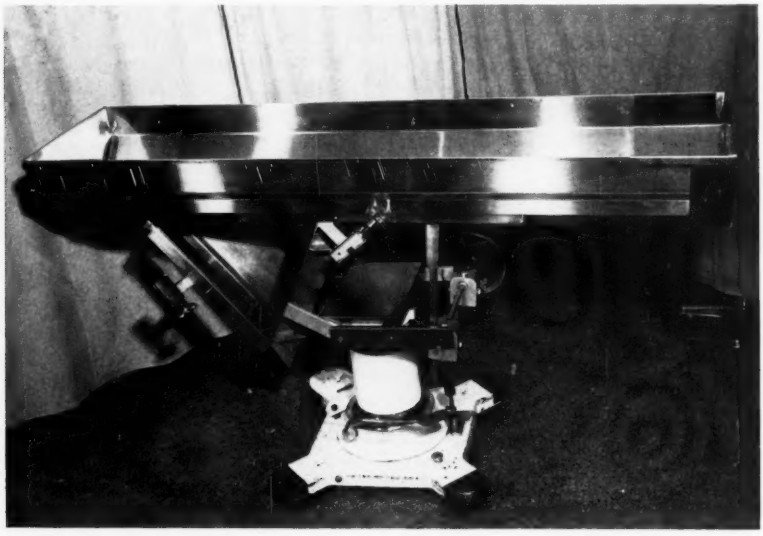

Fig. 1. The refrigerating frame consists of side-panels and end-pieces, which are attached to the operating table after the patient has been placed in position. The trough is rendered watertight by lining it with a sheet of plastic material.*

frame consisting of side-panels and end-pieces which encloses the top of the operating table (Fig. 1). The resulting trough is rendered watertight by lining it with a sheet of waterproof material.

To employ this method, the operating table is draped with the waterproof sheet, and the patient is placed upon it and anesthetized with Thiopental sodium. The

* The combined operating chair and table shown above is described in the following article: Gardner, W. J. A neurosurgical chair, *J. Neurosurg.*, 1955, *12:* 81–86.

frame is then placed in position, its inner surface is lined with the sheet, and the trough is filled with ice water. The patient's head is sufficiently elevated so that the mouth and nose remain above the level of the water. In this bath, the temperature of the body may be lowered to 86°F. (30°C.) within 30 minutes. When the temperature has reached the desired level, the water is removed by siphoning through a garden hose. The trough is then converted to an operating table by removing the frame, and the patient is in position for operation. When the operation is concluded the table may be reconverted into a trough, and the patient immersed in warm water to restore his body temperature before returning him to his bed. The time required for assembling and disassembling the refrigerating trough is about one minute.

NOTICES

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Examinations were given by the American Board of Neurological Surgery in New Haven, Connecticut, October 24–26, 1955, and the following were certified:

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AMERICAN ACADEMY OF NEUROLOGICAL SURGERY

At the 17th annual meeting of the American Academy of Neurological Surgery at Hot Springs, Virginia, October 27–29, 1955, the following officers were elected for the year 1955–1956: President, Dr. Stuart Rowe, Pittsburgh, Pa.; Vice-President, Dr. Jess D. Herrmann, Oklahoma City, Okla.; Secretary-Treasurer, Dr. Eben Alexander, Jr., Winston-Salem, N. C.

The next meeting will be held at the Camelback Inn, Phoenix, Ariz., October 31–November 3, 1956. The second annual American Academy of Neurological Surgery Award will be presented at the 1956 meeting, and the paper to be given at that time will be selected by a committee headed by Dr. Edwin B. Boldrey, San Francisco, Cal.

ASSOCIATION FOR RESEARCH IN NERVOUS AND MENTAL DISEASE

At the 35th annual meeting of the Association for Research in Nervous and Mental Disease held in New York City on December 9 and 10, 1955, the following officers were elected for the year 1956: President, Dr. Harry C. Solomon; First Vice-President, Dr. Stanley Cobb; Second Vice-President, Dr. Wilder Penfield; Secretary-Treasurer, Dr. Rollo J. Masselink; Assistant Secretary, Dr. Lawrence C. Kolb.

SOUTHERN NEUROSURGICAL SOCIETY

The next annual meeting of the Southern Neurosurgical Society will be held in Jacksonville, Fla., March 23 and 24, 1956. Headquarters will be at the George Washington Hotel. The meeting is for members and invited guests.

JOURNAL OF NEUROSURGERY

The *Journal of Neurosurgery* is devoted to the publication of work relating primarily to neurosurgery, including studies in clinical neurophysiology, organic neurology, ophthalmology, radiology and pathology. Short articles on unusual cases and on technique, special instruments or equipment that might be useful to others in the field are also acceptable.

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REFERENCES should be arranged alphabetically and cited by superior numerals in the text. The following order should be used:

Articles: Name of author, full title of article, name of periodical, year, volume, first and last page of article. Abbrevations of names of periodicals should conform to the style used in *A world list of scientific periodicals published in the years 1900–1933* (Oxford University Press, 1934).

Books: Name of author, full title of book, city, publisher, year, edition, full pagination (if book is in more than one volume, give the number of volumes).

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